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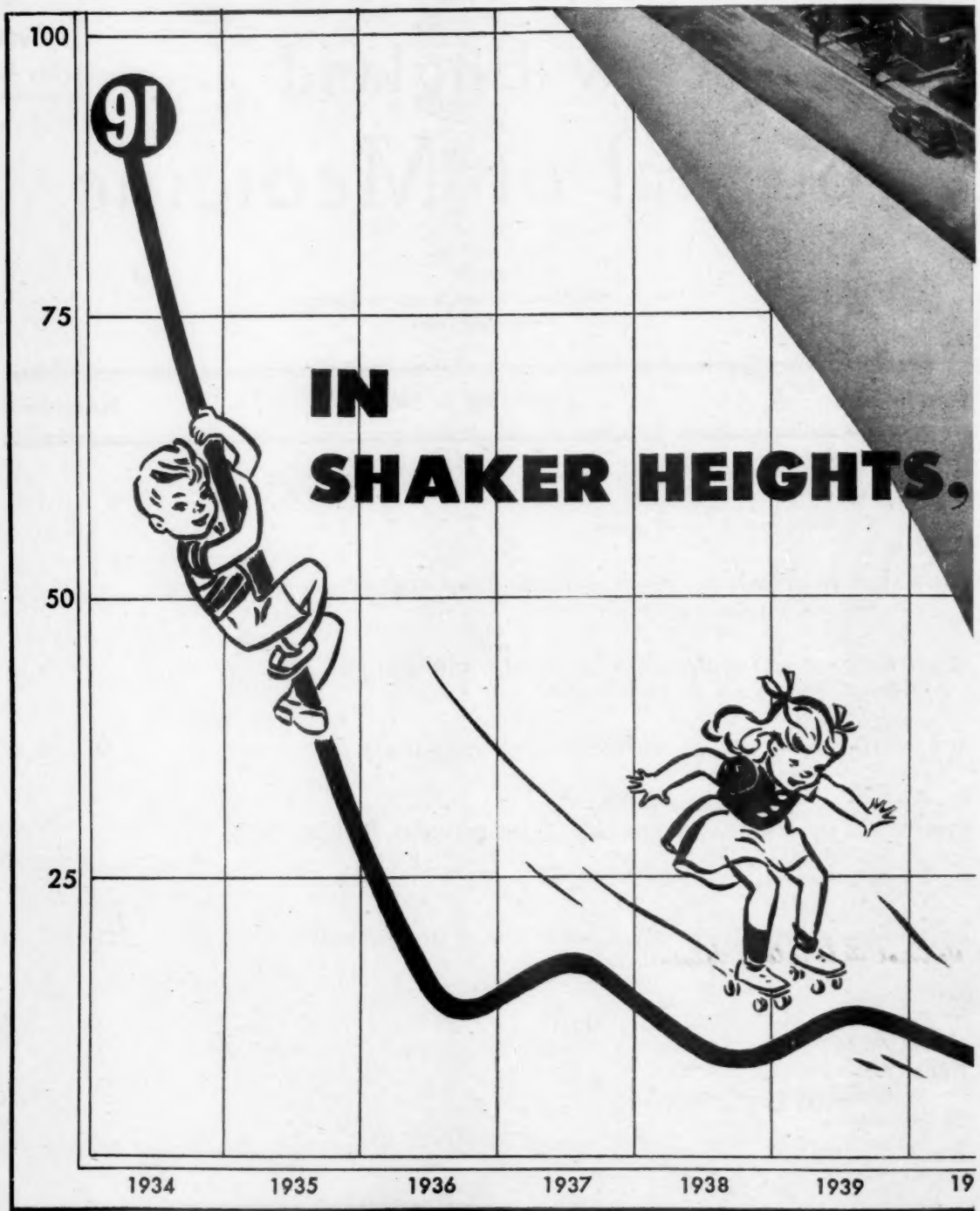

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PUTRID EMPYEMA

JOHN W. STRIEDER, M.D.,* AND JOSEPH P. LYNCH, M.D.†

BOSTON

PUTRID empyema has been recognized as a clinical entity for many years and has recently been the subject of a number of excellent reports.[1–4] Nevertheless, many surgeons fail to distinguish it from ordinary pyogenic empyema and, persisting in this misapprehension, treat it as they ordinarily treat this relatively benign lesion, with disastrous results. It is our belief that with a better understanding of the nature of the disease recognition will be prompter, treatment will be more adequate, and the mortality, which has been high, will be lowered.

The report that follows is based on an analysis of patients on the various services at the Boston City Hospital from June, 1934, to January, 1941, and of those on the Thoracic Surgery Service from January, 1938, to January, 1941 — 90 cases in all. A review of the etiology, bacteriology, pathology and treatment demonstrates that this disease differs strikingly from postpneumonic pneumococcal empyema and other pyogenic empyemas, with which it is so often confused. Furthermore, from the same review it is believed that the advantages of early and radical surgery can be demonstrated.

DEFINITION

Putrid empyema may be briefly described as a sloughing, gangrenous pleuritis caused by certain anaerobic bacteria acting in symbiosis. Its distinctive characteristic is the presence of extremely foul purulent fluid in the pleural cavity, which gives rise to its name. In almost 100 per cent of these patients the fluid obtained by thoracentesis was described by the operator as foul, stinking, sulfurous or putrid. On the other hand, the pleural fluid from no other patient with empyema was so described, in spite of the fact that a number of cases were due to the colon bacillus. The odor is indeed truly distinctive.

It might be more accurate to call the disease "anaerobic empyema," as has been suggested by Maier and Grace,[1] because, from our own studies and those of others, it appears that putrid empyema is always caused by anaerobic organisms. Meleney[5] states that anaerobic infections are usually foul-smelling. When this is the case, one can be almost certain that he is dealing with anaerobic infection and not, as is so often erroneously inferred, with infection by the colon bacillus, which produces little if any odor. One can be certain that if the odor is foul the anaerobic infection is active; but if it is not, the presence of anaerobes cannot be ruled out. Since, however, there is still disagreement about the bacteriology and since the descriptive name is so distinctive, it seems better to continue the name "putrid empyema" until complete proof of the nature of the infection is at hand.

INCIDENCE

The number of cases of putrid empyema in any series of patients suffering from empyema varies with certain factors. Our figure is considerably higher than that found in other series, but this is thought to be a corollary of the high percentage of putrid pulmonary infections, such as lung abscess, bronchiectasis, fusospirochetal pneumonitis and pulmonary gangrene, encountered in a large municipal hospital where the economic and hygienic status of the patients is poor. Neuhof and Hirshfeld[6] found the disease in 17 (9 per cent) of 184 children with empyema. In the present series, covering seven and a half years, 530 patients with acute empyema were treated at the Boston City Hospital, and of these, 90 (17 per cent) had putrid empyema. In general, it appears that 5 to 10 per cent of all cases of empyema are found to be putrid.[7]

That the disease is serious can be appreciated from mortality figures. Neuhof and Hirshfeld[2] have reported a mortality of 47 per cent, and Flack[3] one of 66 per cent. In the series of 57 patients reported herein, which were treated in this hospital but not on the Thoracic Surgery Service, most of them before 1938, the mortality was 54 per cent. These patients were treated on general surgical services by methods that would usually have been adequate for postpneumonic empyema but which,

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according to our criteria, were inadequate for putrid empyema.

Etiology

Generally putrid empyema is secondary to some type of anaerobic infection of the lung. Most frequently it complicates a lung abscess or a gangrenous (fusospirochetal) pneumonitis. In Table 1 are listed the primary diseases in this series, with the mortality rate of the putrid empyema that developed from them.

Table 1. *Relation of Primary Disease to Mortality.*

Primary Disease	No. of Cases	Recoveries	Deaths	Mortality
				%
Lung abscess	35	18	17	49
Bronchiectasis	12	11	1	8
Tuberculosis	6	2	4	67
Pneumonitis (gangrenous)	24	13	10	43 (23 cases*)
Others (including carcinoma of the lung, 1; trauma, 2; and unknown, 10)	13	6	7	54
Totals	90	50	39	
Average				44

*One patient left against advice and the outcome is unknown.

Graham et al.[4] and Rosenblatt[8] state that empyema is one of the most frequent complications of lung abscess. Neuhof and Hirshfeld[2] found that 25 of 104 patients with subacute or chronic lung abscesses developed putrid empyema. Neuhof and his associates[9] also mention this type of empyema as a frequent and serious complication of the surgical drainage of lung abscess. Graham has expressed the belief, with which certain others have agreed, that frequently a lung abscess that has been complicated by putrid empyema heals when the empyema has been adequately drained. Neuhof and his coworkers[9] have denied this, believing that the abscess should be drained at the time of operation for the empyema. In our opinion, spontaneous cures of abscesses depend on the size of the perforation and whether or not drainage resultant on the rupture is adequate. It is our experience that any decision concerning drainage of the abscess at the time of thoracotomy must be taken on the basis of the location of the abscess and the condition of the patient, as will be discussed later.

Graham et al.[4] also state that empyema is a not infrequent complication of bronchiectasis. In the postoperative course of lobectomy for bronchiectasis such an empyema has been an all-too-frequent complication. Most of our cases in this group occurred in the course of an acute exacerbation of the bronchiectasis, and might more properly be considered complications of pneumonitis. In 2 of these, a definite cortical abscess could be seen at operation, and undoubtedly rupture of similar abscesses is the mechanism of empyema formation in many of these patients. It is interesting that, as shown in Table 1, when the putrid empyema complicates bronchiectasis the mortality is lower, out of all proportion, than that of the other groups. The significance of this is conjectural, but may depend on the degree of immunity to the organisms that the patient has built up over a long period through his chronic disease, together with the tendency to localization and stabilization caused by pre-existing pleural adhesions.

Pneumonitis

Pneumonitis is so designated to distinguish it from pneumococcal pneumonia. These cases are most difficult to understand. Only 3 cases in our personal series fall definitely in this group, but some of them may have been the typical pulmonary gangrene that is included in this category. One of our patients was such a case. Another developed as a complication of fusospirochetal pneumonia, which has been described by Peirce and Field,[10] and others were undoubtedly acute exacerbations of pneumonitis on an underlying bronchiectasis. Careful follow-up studies might have revealed this diagnosis in more cases. Eleven of the 12 patients with bronchiectasis were studied on the Thoracic Surgery Service, which suggests that the diagnosis will be made oftener if its possibility is entertained. In general, the onset of the disease in these patients resembled that of pneumonia, yet its chronicity made this diagnosis untenable. In no case were pneumococci found in the sputum, and roentgenograms of all but 3 demonstrated pleural fluid. The diagnosis in most of these patients was based on the history, for in all but 3, even the physical signs on admission were those of fluid. On the basis of our clinical experience, we believe that the acute stage of all these putrid pulmonary infections is a pneumonitis.

The patients who developed this disease as a complication of tuberculosis probably had secondarily infected blocked cavities that ruptured into the pleura.

The remaining patients include 1 who developed pulmonary gangrene distal to an obstructing carcinoma and 2 who developed it after trauma. In one of the latter it followed the instrumental rupture of a bronchus, the patient having had bronchiectasis secondary to a long-standing obstructing foreign body, for the removal of which bronchoscopy was performed. Thirty-six hours following bronchoscopy he became moribund because of unrecognized tension pneumothorax and anaerobic pleural infection, so that only a closed thoracotomy could be done. He continued to grow worse, and died twenty-four hours later. The other patient, whose case we[11] have previously reported, developed a *Clostridium welchii* infection of the pleura following a stab wound in the chest. This complication, seldom seen in civil life, is frequent in wounds of the chest in wartime.

The unknown group includes 10 patients, 3 of whom died, no autopsies being done. The others received no studies except routine roentgenograms.

Bronchograms would undoubtedly have shown bronchiectasis in some cases. Pilot[12] suggests the mechanism in such cases by assuming the formation of an abscess so close to the pleura that rupture occurs before pulmonary destruction is extensive enough to be apparent clinically.

Bacteriology

The bacteriology of putrid empyema is complicated and uncertain, mainly because of two factors. First, the fact that the infection is due to anaerobic bacteria has prevented complete and accurate correlation of the clinical and bacteriologic studies. Only during the last ten to fifteen years has much study been devoted to these organisms, and even at the present time relatively few hospitals are adequately equipped to do complete anaerobic studies, as pointed out by Meleney.[13] Without such technical facilities the diagnosis cannot be made, for the general experience has been that anaerobic cultures are needed. In our experience, repeated routine cultures of pleural fluid not infrequently show no growth, but the anaerobic culture demonstrates the presence of infection. Second, it seems to be established that the problem of symbiosis is involved in putrid empyema and its related, and usually antecedent, diseases. So general has been the belief that one organism must cause each disease and that Koch's postulates must be satisfied that the work on infections due to the synergistic action of two or more organisms has been retarded. That certain organisms favor the growth and metabolic activity of other species in the same surroundings is well known. It has also been demonstrated that in the development of certain pathologic processes by mixed cultures one species may initiate the inflammatory reaction in the host and the other may complete it. This conception has been suggested by Altemeier,[14] who concludes that although a great deal of work has been done, much remains to be accomplished.

Leeuwenhoek in 1683 first described the fusiform bacillus and the spirochete,[15] and Laennec in 1819 described the first case of pulmonary gangrene. Longacre and Herrmann[16] have discussed the work of Leyden and Jaffe, who in 1867 first associated the spirochete with putrid bronchiectasis and pulmonary abscess; and Kline and Berger[17] mention Plaut (1894) and Vincent (1896) as having identified these organisms and named them as the cause of the mouth infection now known as Vincent's angina.

A review by Fisher and Abernethy[18] states that in 1893 Veillon mentioned the anaerobic streptococcus, which he had isolated from a foul bartholinitis. Since that time this organism has most frequently been isolated from foul lesions of the lung and pleura and from puerperal infections. These authors credit Rendu and Rist (1899) with the description of the association of the anaerobic streptococcus with the gram-negative bacillus.

Until World War I, however, the anaerobes received little attention, but following the armistice much work was done on them. The anaerobic spore-bearing gram-positive bacilli received the greatest attention and consequently are the best understood. Some of those may cause putrid empyema, usually following trauma, but this seldom occurs in civil life.[11, 19] The gram-positive cocci have been recognized, and many attempts to classify them have been made, but much study is still needed. Meleney and his co-workers have been most successful with their researches into microaerophilic streptococci. The gram-negative and gram-positive non-sporulating bacilli have received the least attention of all. In 1905, Weaver and Tunnicliff,[20] after successfully culturing the fusiform bacillus, were unable to produce any pathologic lesions with the pure culture; yet when a mixed culture was used, a gangrenous infection resulted. This inability to produce disease with the pure culture of these organisms gave rise to the impression that they were mere saprophytes, and many still hold this impression.[21] Smith[22] believes that they are not secondary saprophytic invaders. In 150 patients with pulmonary tuberculosis, 6 with asthma and 5 with mycotic lung infections, the fusiform bacilli and the spirochetes were absent in every case, despite repeated examinations. If they were secondary invaders, one would expect them to be present in almost every form of chronic pulmonary disease.

Despite all this work, up to 1928 only about 150 cases of pulmonary fusospirochetal infection had been reported. After this, however, the number of reports relating these organisms to pulmonary gangrene, lung abscess and bronchiectasis greatly increased. Moreover, the excellent work of Smith,[22] Kline[23] and Pilot and Davis[24] has clearly shown that these anaerobes in association — that is, the organisms usually found in Vincent's angina, peridental infection and pyorrhea alveolaris — are the same as those usually found in putrid lung infections, and that these infections may be produced with them. Smith[25] has reported cases in which contact with a case of Vincent's infection resulted in pulmonary fusospirochetosis and that exactly the same organisms were found in both diseases. More will be said about this in connection with the experimental production of these diseases.

It should be remembered, as has been emphasized by Smith[26] and others, that although these infections are usually referred to as fusospirochetal, this is mainly because these organisms are so easily identified on smear. The anaerobic cocci are always associated with them and are of equal importance.

As revealed in the literature, the bacteriology of putrid empyema seems complex, each author emphasizing a different organism or group of organisms. Fisher and Abernethy[18] stressed the role of the

anaerobic streptococcus, which they were able to find in all the 7 cases they reported. Flack[3] reported 3 cases in which the fusospirochetal organisms seemed to be the important factor. Kline and Berger[27] and Farbman and Danforth[28] have also reported cases in which the fusospirochetal organisms were observed, and the former are especially convinced that these are the pathogenic agent. Maier and Grace[1] describe the cause as a mixed bacterial flora, with the fusiform bacilli, spirochetes and the anaerobic streptococcus those most frequently found. They believe, furthermore, that symbiosis is an important factor in the action fo these organisms. Sauerbruch and O'Shaughnessy[29] also name the anaerobes as the cause of putrid empyema.

In Table 2 are presented the results of the bacteriologic findings in this series. The routine procedure now adopted by the Bacteriology Department is to culture all empyema fluids under anaerobic conditions (cooked-meat medium), as well as aerobically (blood broth and blood-agar plates). Stained smears serve to identify morphologically the fusiform bacilli and spirochetes, and these occasionally grow in the cooked-meat medium. The strictly anaerobic streptococci also grow in the cooked-meat medium and can be subcultured to blood-agar plates that are incubated under anaerobic conditions. The facultative anaerobic streptococci grow both in the cooked-meat medium and in the blood broth, and on subculture will grow on blood-agar plates incubated under ordinary atmospheric conditions, although much better growth is obtained if the plates are incubated in an atmosphere containing an increased amount of carbon dioxide (Nye and Lamb[30]). Most strains of the last organism are beta hemolytic on blood agar and they are closely related to, if not identical with, the microaerophilic streptococci described by Meleney.[13]

TABLE 2. *Bacteriological Data.*

ORGANISM	NO. OF CASES	RECOVERIES	DEATHS	MORTALITY
				%
Fusiform bacilli (alone) ..	18	9	9	50
Streptococcus (strict anaerobe)	8	5	3	38
Streptococcus (facultative anaerobe)	63	35	27	44 (62 cases)
Cl. welchii	1	1	0	0
Totals	90	50	39	

The fusiform bacillus was identified in every patient except the one in whom the infecting organism was *Clostridium welchii*. In only 9 patients were aerobic organisms found, and never were they present without an anaerobe — this in spite of the fact that aerobic cultures were made in every case. These aerobes were the streptococcus, pneumococcus and staphylococcus and the influenza bacillus. Again, it should be pointed out that if aerobic cultures alone were made on all cases, the true nature of the disease might have been overlooked, and the assumption might have been made that the empyema was due to the aerobic organism.

The fusiform bacillus has been described as a strict anaerobe, and many different types have been identified by their morphology alone. Some attempts have been made to distinguish them serologically. The spirochetes have proved to be the most difficult to grow, but they were reported as seen on the direct smear in one quarter of our cases. Four types of the spirochete have been identified by Smith.[26]

PATHOLOGY

Putrid empyema presents itself as a gangrenous, hemorrhagic pleuritis, with the production of a large amount of fluid and, on occasion, gas. It complicates a gangrenous process of the lung that may be acute or chronic. When the parent disease is acute, there is usually total involvement of the pleura, but when the underlying condition has existed for some time, a moderate amount of walling off may have occurred, and a localized type of empyema then develops. Tissue necrosis is, however, one of the outstanding features of anaerobic infections; hence, the adhesions tend to disappear, and there may result one or more large or small compartments filled with foul pus and débris. The formation of fibrinous adhesions occurs early in every case, and this is an important feature in the treatment of these patients.

The fluid in this disease tends to be thin for a long time, but the pleural reaction is so marked that the mediastinum becomes fixed early.[1] In this series of 90 patients, the fluid was described as thin in 45, creamy in 13 and thick in 24, and was not mentioned in 8. Some patients had a clear serous fluid at the first thoracentesis. Smith[26] has also described this, noting that the fluid may be clear for the first day or so. In the present series, the culture of the clear fluid often showed the anaerobes and the characteristic foul odor was noted. In other cases the fluid was odorless. In 19 cases, the pleural fluid appeared clear on the first thoracentesis, but a second thoracentesis, soon after the first, produced fluid that was both foul and purulent.

The presence of a bronchopleural fistula has frequently been noted in patients with this disease. Its occurrence depends on the underlying disease. The incidence is high in empyema complicating the rupture of a lung abscess; it is medium in the bronchiectasis group and low in the pneumonitis group. In this series a fistula was noted in 25 patients, and may have been present in others, especially in the 19 not operated on. It caused little change in the course of the disease, however, and in all cases closed before there was complete obliteration of the empyema cavity. In 20 cases the fistulas were in the abscess and bronchiectasis groups, and in 2 they were in the tuberculous group.

ANIMAL EXPERIMENTS

The bacteriologic investigation of putrid empyema by experimental methods has been neglected, but

many investigations of the primary disease have been made, and much of the present discussion concerns these. Pilot and Davis[24] were able to show that a pure culture of fusiform bacilli had little pathogenicity. In the pleural cavity the organisms caused only a slight fibrinous exudate. The cocci — 80 per cent of which were anaerobic streptococci — alone produced a serofibrinopurulent pleuritis without odor. When the spirochetes, fusiform bacilli and cocci were combined, however, the lesion had a putrid odor. Smith[7] has produced pulmonary abscesses, bronchiectasis, gangrene and pneumonia by the intratracheal injection of material from the alveolar border of patients with pyorrhea. He demonstrated that four organisms, the anaerobic streptococcus, the fusiform bacillus, a spirochete and a vibrio, were needed. None of these alone, nor any mixture without all of them, produced the characteristic lesions.

Many others[5, 16, 23, 31–36] have been able to produce these lesions by using the anaerobes, some by the intratracheal route and others by means of infected emboli. The material used was generally obtained from the scrapings of the teeth of patients with pyorrhea alveolaris, and was usually shown to contain the anaerobes mentioned above. Crowe and Scarff[34] point out the many unsuccessful attempts to produce lung abscesses by the intratracheal inoculation of virulent forms of staphylococcus, streptococcus, pneumococcus and the colon bacillus. Yet when the anaerobes were employed, the usual result was a localized chronic pulmonary suppuration.

The incidence of empyema is not mentioned in most of the results reported. Longacre and Herrmann,[16] however, give the incidence in their experimental series as 10 per cent following the intratracheal method and 33 per cent following the use of emboli infected with anaerobes.

Method of Infection

Almost all writers agree that there is infection about the mouth in these patients. Usually there is pyorrhea alveolaris, chronic ulceration of the mouth or a Vincent's infection. Meleney[5] notes that the most frequent anaerobic infections are those about the mouth. These organisms may be aspirated, with resulting pneumonitis or abscess and the complication of putrid empyema. Duffy[37] was able to show fusospirochetes in all his patients. Stern[38] concludes that a thorough investigation of the dental history in cases of abscess of the lung may reduce the percentage of cases formerly grouped as pulmonary abscess of unknown origin. He was chiefly concerned with antecedent dental operations, but we have been greatly impressed by the high incidence of unhygienic mouths encountered in patients with putrid empyema without a history of any dental procedure. Thus, the taking of alcohol, poor oral hygiene and a period of unconsciousness prior to the onset of the disease seem to be the usual combination of factors culminating in putrid empyema that is seen in large municipal hospitals. Smith[26] also believes that aspiration of infected material from the mouth probably accounts for most pulmonary abscesses, although a small number may be due to emboli from the respiratory passages. In this series a description of the teeth was present in the records of 70 patients. Of these, the teeth were described as good in 7, as negative in 3 and as showing pyorrhea or chronic infection in 60. This means that at least 85 per cent of the patients had infection about the teeth.

Clinical Features

Certain clinical features are distinctive of putrid empyema. In this series there was a marked predominance of males who comprised 72 of the 90 patients. The disease was, however, about equally fatal in both sexes, the mortality being 46 per cent in males and 39 per cent in females.

The ages of the patients varied from five to seventy-eight years. The relation between age and mortality is shown in Table 3. In general, mortality increased with age. An explanation for the low mortality in the sixty-to-sixty-nine-year group is suggested by the type of surgery employed. In this group, 5 of 7 patients recovered and all of these, and only these, had adequate surgery. This indicates that the other patients in the older age groups were deprived of the chance that adequate surgery might have offered. In many cases the records showed that there had been much waiting for the patient's condition to improve, whereas it deteriorated rapidly and progressively. The condition on admission was said to be poor in 42 patients, fair in 27 and good in 15. The condition of 6 patients was not recorded. In other words, most of the patients appeared to be acutely ill at the time of admission. Their poor general condition is not an accurate indication for delaying surgery; on the contrary, it suggests that immediate surgery is imperative.

Table 3. *Relation of Age and Mortality.*

Age	No. of Cases	Recoveries	Deaths	Mortality
yr.				%
1– 9	4	4	0	0
10–19	11	9	2	18
20–29	12	8	4	33
30–39	18	10	8	44
40–49	23	11	11	50 (22 cases)
50–59	9	3	6	67
60–69	7	5	2	29
70 up	6	0	6	100
Totals	90	50	39	

Although the disease evolves in a variety of ways, there are some constant signs. A frequent complaint is severe pain, often localized, and persisting even after the development of fluid. Associated with this is a localized tenderness of the chest wall, which is a frequent indication of severe gangrenous

infections of the pleura. The clinical impression on admission in many patients was that of pneumonia, usually based on a history of pain in the chest, chills and cough; yet the physical signs were those of fluid, which characteristically develops early in the disease.[2] The roentgenogram was occasionally misleading, showing consolidation even when fluid was present. A history of cough of long duration, often with foul sputum, was frequent and was helpful in making the diagnosis.

These patients are often far more ill than one would expect from the amount of discoverable disease. They early become toxic and delirious. They frequently show signs of peripheral vascular collapse, with cold, clammy extremities and an extremely rapid pulse. The temperature, although elevated, is not in proportion to the pulse, a feature common to all anaerobic infections.

The diagnosis of this disease is made by thoracentesis. Fluid having a foul odor is pathognomonic, although a direct smear that reveals the fusospirochetes is corroborative evidence, easily obtained. Anaerobic culture of the fluid must be done, for aerobic culture does not reveal the true nature of the infection. In reviewing these cases, it was not unusual to find that two or more aerobic cultures had been done without growth before an anaerobic culture revealed the infecting organisms. Purulent chest fluids should always be cultured anaerobically, especially those that are foul.

One complication of thoracentesis, cellulitis of the chest wall, has been noted by many authors.[1, 2, 17, 18] It is so grave that every precaution should be taken to circumvent its occurrence. Apparently the thin, watery fluid easily passes along the needle track into the deep tissues of the chest wall. There, under the muscles, in the tissues overlying the ribs, conditions are ideal for the growth of anaerobes. Severe pain and tenderness soon appear. Within twenty-four hours there is marked redness, and a cellulitis with gangrene of the deep tissue rapidly develops. Because of this, most observers have wisely stated that thoracentesis should be followed by surgery as soon as possible and that repeated thoracenteses are contraindicated. It is true that in some patients thoracentesis can be done more than once without this complication; on the other hand, when it develops it is of such severity that it approaches in importance or overshadows the underlying disease.

Maier and Grace[1] encountered cellulitis of the chest wall in 4 of their 23 patients, despite the fact that about half the patients were operated on within twenty-four hours of the thoracentesis. In the present series, 21 patients developed this complication. If those operated on within twenty-four hours are eliminated, 38 per cent developed it. In our experience this complication is so severe and frequent that we, too, believe that thoracentesis should be done only for diagnostic purposes and should be immediately followed by open drainage. Also, because this complication may develop, closed thoracotomy or the suture of wounds after rib resection is extremely hazardous.

Treatment

The rate of recovery is greatest in the patients operated on early. The average of the time intervals from the onset of the disease — calculated as accurately as possible from the records — to the time of operation in those who lived was 5.2 days, and in those who died it was 10.7 days. This may be expressed in another way by considering all the patients who had surgical treatment. On the first to the third day, 25 patients were operated on and 22 recovered, a mortality of 12 per cent. On the fourth to the seventh day, 22 were operated on and 16 recovered, a mortality of 27 per cent. On the eighth to the tenth day, 13 were operated on, and only 6 recovered, a mortality of 54 per cent. Hence, it can be seen that 75 per cent of the patients who were operated on in the first week of their disease recovered, whereas slightly more than one third of those who died were operated on within the first week. Further evidence of the need for early surgery can be obtained from the fact that of the 18 patients who died without operation, 10 did so within the first ten days of their disease. In 7 cases, it was impossible to calculate the exact duration of the disease with any accuracy, but in most of the cases the course was probably less than ten days. Only 1 patient lived with the disease over the ten-day period.

With few exceptions there is unanimity of opinion on the treatment of this disease. All writers agree that surgery is indicated and that open drainage is the treatment of choice. Graham, Singer and Ballon[4] believe that it is safer to aspirate frequently or to use closed drainage, and give as their reasons those that generally pertain to pyogenic empyema. Flack[3] thinks that conservative treatment with an intercostal tube and later resection of a rib is the treatment of choice. The differences of opinion on the treatment of the disease depend largely on the question of mediastinal stabilization. If one applies the usual principles that govern the treatment of postpneumonic empyema and awaits thickening of the purulent fluid to the consistence at which mediastinal fixation may be postulated, valuable time may be lost.

The sole attribute common to all anaerobes is their inability to multiply or even live in an atmosphere rich in oxygen. It has been shown that they succumb after only a few hours of contact with oxygen, even in low concentration. Consequently, most writers have agreed that open operation with adequate aeration and evacuation of the pus is the best treatment.

Moreover, in this disease, despite the consistence of the fluid, there is prompt stabilization of the

mediastinum, so that early operation may be accomplished. Maier and Grace[1] noted that all except one patient in their series with thin fluid had a fixed mediastinum.

In 1938, Dolley and Jones[39] described a method for open drainage of acute pyopneumothorax in patients in whom the mediastinum is not stabilized. This procedure is simple and logical and has proved most successful in our patients. It is to be remembered that the cases causing concern about the stability of the mediastinum are few, for, as has been noted, early fixation is the rule. The method to be described provides an adequate method of treatment in the event that a mobile mediastinum is encountered. With this method none of our patients developed chronic empyema, and the average time of treatment from operation to complete closure of the wound was seven weeks. Another type of treatment, which agrees with ours in principle but differs slightly from it in technic, is that described by Sauerbruch and O'Shaughnessy.[29] This consists in the removal of two dependent ribs under positive-pressure anaesthesia and open packing of the wound.

Operative Technic

The operative method that we have used follows closely that of Dolley and Jones,[39] described for the treatment of acute pyopneumothorax following the rupture of a lung abscess. We have used 1 per cent procaine infiltration as the anesthetic of choice. A 20-cm. incision is made over the most dependent rib, as determined by thoracentesis, and is extended from the posterior to the anterior axillary line. It is carried through the muscles, and a segment of the appropriate rib as long as the incision is resected subperiosteally in the usual fashion. A large dressing has already been prepared for immediate use.

From this point the procedure is determined by the degree of mediastinal fixation. This may be judged by the amount of pleural thickening or by means of a small incision into the pleural cavity. If there is inadequate fixation, the pleura is quickly incised the entire length of the incision, the dressing is applied, and the patient is quickly turned on his back. With this routine rapidly performed, the dangers of open pneumothorax are largely circumvented, for the dressing soon becomes saturated and serves as a tampon, preventing the exchange of air through the wound; yet the wide opening thus secured allows complete drainage into the dressing. The dressing is not changed until the third day, when a quick change is made. Stabilization usually occurs from the fifth to the seventh day, and from then on a tube or pack can be used. It has been our policy to use a gauze pack impregnated with activated zinc peroxide, placed partly in the pleural cavity and partly in the wound. By this means the odor disappears and the wound is clean and granulating five to seven days after the treatment is begun. Tubes are then inserted. The convalescent treatment is the same as that for the usual postpneumonic empyema. The use of zinc peroxide has been found of value by many others in anaerobic infections,[1, 8, 17, 40] and we cannot emphasize its value too strongly for this type of disease.

If the fixation is adequate, more time can be spent examining the cavity and removing all the pus and fibrin by suction. Otherwise, the treatment is the same, except that the zinc peroxide is used from the time of operation.

Under no circumstances is an attempt made at any type of wound approximation, and hemostasis is obtained with a minimum of ligatures.

Most of the patients so treated show immediate improvement. In a few days the appetite returns and they become more active in bed. For a considerable period, often two or three weeks, however, these patients have a low-grade fever. apparently due to the primary intrapulmonary infection. Irrigations are not always possible because of the existence of a fistula. If one is not present, irrigations may be helpful because of their mechanical cleansing action.

Drug Treatment

The effect of the sulfonamides on the anaerobes is still uncertain. Some evidence has appeared that these drugs are of no value in this type of infection, but this is confusing and uncertain. Meleney[5] believes that their value in this type of infection, in which there is early destruction of tissue, has not been demonstrated. Maier and Grace[1] found that the typical dramatic response to these drugs did not occur in this type of infection. In fact, they believe that this failure of response is a point of differentiation between the atypical pneumonia associated with foul empyema and the typical pneumococcal pneumonia.

Twenty-one of the patients in this series were treated with one of the sulfonamides — usually sulfathiazole or sulfapyridine. Of these, only 1, with an infection due to *Cl. welchii*, showed a favorable response. We are of the opinion that these drugs are of no benefit once the empyema has developed, regardless of what value they may prove to have for the primary disease. The other drugs ordinarily used in fusospirochetal infections are the arsenicals. Despite any questionable value they may have for the primary disease, they are of no worth in the treatment of putrid empyema.

We have treated recently 2 cases of putrid empyema, not included in this series, by the intramuscular and intrapleural injection of penicillin. In the first case the empyema complicated a pneumonitis that was apparently the result of the aspiration of infected material from the mouth. The pleural fluid was typically putrid, and cultures re-

vealed the usual mixed flora. Twenty-five thousand units of penicillin was daily administered intrapleurally for ten days, a total of 250,000 units. Following this the fever persisted, and five days later rib resection was performed, with the intention of draining a residual pocket. Exploration revealed only fresh adhesions and a thickened pleura. The chest was closed without drainage, and the patient made an uneventful recovery.

In the second case the empyema complicated a pneumonitis that resulted from the post-partum aspiration of vomitus or mouth secretions or both. The patient was considered moribund when first observed. She received 10,000 to 60,000 units of penicillin intrapleurally daily for fourteen days, a total of 368,000 units. During that time there was striking improvement in the general condition. On the fifteenth day, rib resection was performed for the drainage of a localized empyema. The patient also received an average of 120,000 units of penicillin intramuscularly daily for thirty-nine days, a total of 4,640,000 units. A lung abscess that developed in the upper lobe at the site of the original pneumonitis was drained in two stages on the thirtieth and thirty-sixth days. The patient was discharged on the seventy-fourth day, the temperature having been normal for fifteen days. She has been well for over a year.

One may conclude that penicillin is a valuable adjunct in the treatment of putrid empyema and effects cures in a certain percentage of cases.

Results

Tables 4 and 5 show the results of treatment in this series. The former includes the total series of patients, whereas the latter records only those who were treated on the Thoracic Surgery Service. In explanation of these tables, "rib resection" refers to the usual type of operation done for empyema,

Table 4. *Results in the Total Series.*

Type of Treatment	No. of Cases	Recoveries	Deaths	Mortality
				%
No operation	19	0	18	100 (18 cases)
Closed thoracotomy	13	2	11	85
Rib resection	31	25	6	19
Open resection	22	19	3	14
Combined closed and open thoracotomy	5	4	1	20
Totals	90	50	39	

with wound closure about one or more tubes. "Open resection" refers to the method described herein, with wound left widely open.

If one first considers the mortality for each type of operation, one sees clearly the value of the adequate types of surgery as represented by rib resection and open resection. The results with the conventional rib resection were good, and the insertion of tubes may be helpful, if there is time. We believe, however, that the use of zinc peroxide is specifically so valuable that it is better to wait until the anaerobic infection has cleared, as judged by the foul odor, before using the tubes. Finally, closure of the wound is of no value and may cause definite harm in some patients, for in this series one third of the patients whose wound was sutured developed a gangrenous, sloughing wound infection, with associated necrotizing phlegmon and eventual complete dehiscence.

Table 5. *Results in Patients Treated on the Thoracic Surgery Service.*

Type of Operation	No. of Cases	Recoveries	Deaths	Mortality
				%
Closed thoracotomy	2	0	2	100
Rib resection	9	6	3	33
Open resection	21	18	3	14
Combined closed and open thoracotomy	2	2	0	0
Totals	34	26	8	
Average				23

These results demonstrate the benefit of immediate surgery of the open type. The 2 patients on the Thoracic Surgery Service who had a closed thoracotomy were seen late, when they were suffering the combined effects of overwhelming sepsis and severe tension pneumothorax, with marked mediastinal displacement. The closed thoracotomy was done immediately as a temporizing measure, but the patients died soon after it, before more could be done. It is doubtful that any type of operation would have helped one of these patients who had extensive bilateral pulmonary tuberculosis.

The results of combined surgery — that is, closed and open thoracotomy — appear to be good, but in both patients on the Thoracic Surgery Service the closed drainage represented a temporizing measure done immediately to correct altered chest mechanics and to allow time for general treatment. In both cases, open resection was done very shortly after the tube drainage. In the other 2 recovered patients who had combined surgery in the total series, both had closed drainage for many months, which proved to be inadequate and had to be followed by rib resection for adequate drainage. In 1 case, however, a chronic empyema resulted.

The 3 deaths among the 21 patients receiving open resection indicate that the mortality when treatment is adequate is usually from the primary disease. One patient died from multiple acute lung abscesses, which had been progressing when the empyema developed. The second died from a gangrenous pneumonitis involving the entire left lung and the lower right lobe. The empyema was on the left, and drainage was accomplished without incident. One week after the drainage, however, spread of the intrapulmonary disease to the right side occurred, and the course was downhill thereafter. The third patient, who had extensive intrapulmonary disease, died twenty-two days after the drainage of a brain abscess, although the empyema was healing well.

Summary

Putrid empyema is shown to be a distinct disease entity differing in its etiology, bacteriology, pathology and clinical aspects from postpneumonic or other pyogenic empyemas.

Bacteriologically, the disease is due to certain anaerobes that produce a gangrenous sloughing pleuritis. Clinically, it is characterized when untreated as an acute onset followed by a rapid severe course and an early fatal termination.

The fact is stressed that in many patients the fluid is thin for some time. This characteristic should be noted but should not be considered a criterion in treatment. Concern about the consistence of the fluid is fatal, for it is a frequently noted fact that stabilization of the mediastinum occurs early in these patients. If the mediastinum is not stabilized, patients can be operated on by the method herein described and thereby receive the benefits of early and open operation.

A review of the 90 cases reported demonstrates the value of early open operation. Zinc peroxide is of distinct value in postoperative treatment. Once the foul odor has disappeared and the hazard of open pneumothorax is in abeyance, these patients may be treated like those with the ordinary pyogenic empyema.

Further experience may show that penicillin is a valuable adjunct in the treatment of putrid empyema.

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THE EARLY DIAGNOSIS OF MINIMAL PULMONARY TUBERCULOSIS*

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THERE are numerous vital reasons that indicate the importance of diagnosing tuberculosis in the minimal stage. The results of sanatorium treatment and follow-up analyses many years after discharge have uniformly shown the following constant and unequivocal advantages for minimal cases in comparison with advanced tuberculosis.[1–4] The percentage of arrested or healed cases is highest, and the time required to obtain an arrest of the disease much the shortest in minimal cases. This group also has the smallest incidence of instability. Collapse measures are infrequent and when used are less extensive, with complications, morbidity and mortality thereby reduced. The sputum is not so often positive as in moderately and far advanced cases. This is important, for all therapeutic procedures aim at the reversal of a positive to a negative sputum. The great majority of patients with persistently positive sputums sooner or later progress and have a bad prognosis, with little chance of survival. During the institutional and follow-up phases of treatment, the number of deaths are markedly less in cases diagnosed in the minimal stage than in those found with advanced tuberculosis, and the patients live much longer. The need for patients to be readmitted after discharge from the sanatorium increases as the stage of the disease advances. The minimal cases, moreover, have far fewer recurrences. All follow-up studies also show that the percentage of those employed or able to work is much higher in this group.

There are great benefits to the public in the early diagnosis and cure of the disease. The spread of tuberculosis from persons with positive sputums to their families and friends and the community is avoided, and a health hazard of first importance is controlled. The shorter hospitalization period required for minimal cases means that cure is effected at a lower cost. Furthermore, these patients often adjust themselves economically, and public expenditures for them and their families are thereby definitely lowered.

The practicing physician is an extremely valuable case-finding agent, since he is in a position to discover a vast number of persons with tuberculosis. Indeed, a large portion of patients see him before anyone else. These opportunities for diagnosis are particularly significant at this time, since war, as always, has increased the incidence of tuberculosis.

To determine the factors that could aid the making of an early diagnosis, 200 cases with minimal lesions were studied. These patients were consecutive admissions to the sanatorium, and all the cases were minimal on admission, according to the standards of the National Tuberculosis Association.

Of the entire series, 155 patients were between the ages of fifteen and thirty-five. Forty-five patients were over thirty-five years old, with 20 of them older than forty. There has not been enough emphasis on the fact that tuberculosis often occurs in the elderly. Older people represent a great source of infection, for far too frequently their symptoms are explained on the basis of other conditions, such as a chronic bronchitis, asthma or winter or cigarette cough. The 200 patients were quite evenly distributed according to sex — 96 males and 104 females. One hundred and one were single, 86 were married, and 13 had separated or been divorced. Of the entire group 123 were Whites, 52 were Negroes, 5 were Chinese, and 1 was a Filipino. Over forty different occupations were represented. These personal data indicate that tuberculosis should be suspected at any age and in any occupation. They may also have any type of social or economic background.

One hundred and thirty-three patients had no specific contact history. Fifty-seven had had definite close association with tuberculosis, 7 had had slight contact, and 3 had had possible exposure. In other words, 67 patients (34 per cent) had had some contact with tuberculosis.

A history of contact with this disease is significant mainly in its indication that the patient has had opportunities to become infected. In fact, many more cases are found in these persons than in the general population. The high tuberculosis morbidity and mortality rate for those with a history of household contact has often been emphasized and the particular danger for children has also been stressed.[5, 6] Every person who has had contact with tuberculous patients should have a complete examination, including x-ray, at once. This should be repeated every year, and preferably every six months. As our figures show, however, lack of contact does not rule out the presence of tuberculosis.

Of these 200 patients, only 37 (19 per cent), were without symptoms. One hundred and fifty-five patients had specific complaints, and 8 gave a history of pleurisy. All the patients had symptoms before or at the time of diagnosis except 6 who developed them after diagnosis. Table 1 lists the symptoms and the number of patients in whom they were present. These symptoms may also be classified according to the body system involved. Pulmonary symptoms comprised 44 per cent, con-

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stitutional or general symptoms, 36 per cent, pleural symptoms 12 per cent, gastrointestinal symptoms 4 per cent, and laryngeal, bone and joint, nervous system, genitourinary, nasopharyngeal and lymphatic system each 1 per cent or less.

The statement has often been made that minimal tuberculosis is asymptomatic. This study significantly shows that the great majority of these patients

TABLE 1. *Distribution of Symptoms.**

SYMPTOM	No. OF CASES
Cough	98
Expectoration	70
Loss of weight	68
Chest pain	61
Weakness (fatigue or lassitude)	55
Fever	28
Hemoptysis	24
Night sweats	24
Streaking	18
Dyspnea	15
Anorexia	13
Chills	9
Hoarseness	6
Irritability	3
Low-back pain	3
Joint pain	3
Diarrhea	2
Headache	2
Insomnia	2

*There were single cases of the following complaints: amenorrhea, polyuria, cervical-node enlargement, nasal discharge, sore throat, spine pain, constipation, dysphagia, nausea, abdominal pain, melena, rectal-sinus discharge, dizziness, laryngeal pain and orthopnea.

do have symptoms. Practically all the symptoms in our patients were unquestionably associated with the tuberculosis. It may be argued that sanatorium patients are symptom-conscious. We believe that complaints actually exist, and a careful history and close questioning of the patients will verify this fact. Frequently only after a diagnosis has been made by routine roentgenography does the patient realize that he has symptoms. This is because the symptoms are not often those of a startling character, such as hemoptysis, blood-streaking of the sputum or pleural pain, but instead are those not arresting in nature, such as cough, — usually explained as due to cigarettes, — fatigue or slight loss of weight. The patients usually consider these of minor importance. These beliefs are substantiated by the experience in the 91 patients in whom a diagnosis was made by routine examination (90 by x-ray and 1 by fluoroscopy), 55 (60 per cent) of whom had symptoms. Abeles and Pinner[7] observed 91 patients whose tuberculosis was accidentally discovered on routine examination. Of this group, 28 cases were minimal, and 89 per cent of these patients had had symptoms at some time.

Certain symptoms are particularly frequent in minimal tuberculosis. Unfortunately, however, these are not characteristic or specific for this disease alone. They may be present in nontuberculous lung or pleural lesions or even in nonpulmonary conditions. Symptoms in themselves, therefore, cannot serve as the sole criterion for the diagnosis of tuberculosis, but when present they should definitely suggest the disease, and the diagnosis can then be made only by following through with a chest x-ray examination.

Besides contact and routine examinations, the only factor that aided early diagnosis in this series was an acute onset or hemoptysis that prompted an immediate medical checkup. On the other hand, delays in diagnosis were seen because of noncooperativeness in the patients. This was usually due to their failure to seek medical advice early, but occasionally a contact neglected to undergo the examination recommended by the physician. There is much need for education of the public to consult physicians early regarding symptoms. The importance of the symptoms should be stressed, especially those that are not striking in character, which have too often been considered trivial.

In 21 patients a diagnosis of tuberculosis was not suspected or made at the first medical consultation or examination. If the 91 patients diagnosed routinely are not considered, this means that 21 of 109 patients (19 per cent) were missed. The mistaken diagnoses included the following conditions: pneumonia, 3 cases; pleurisy, 3 cases; asthma, 3 cases; "cold," tonsillitis, and dyspepsia and colitis, 2 cases each; and influenza, sinusitis, nephritis, rheumatic heart disease, arthritis and unexplained fever, 1 case each.

Almost 75 per cent of the errors were in diagnoses of nontuberculous respiratory or upper respiratory conditions. To prevent these errors, it should be a routine practice not to make a diagnosis of pneumonia, bronchitis, asthma, pleurisy, chest cold, catarrhal fever or grippe without first considering the possibility of tuberculosis. The symptoms and physical findings in these cases may be the same as those encountered in tuberculosis. To determine definitely the presence of tuberculosis an x-ray and sputum examination and occasionally a tuberculin test are requisite for all these patients. This would not be an impractical or uneconomical procedure, for the yield of active cases of tuberculosis would be considerable.

The most valuable diagnostic physical finding in tuberculosis is the presence of rales. Changes in fremitus, dullness and breath sounds are not significant enough to be considered. Only 57 (29 per cent) of these 200 minimal cases had rales. This shows how unreliable a physical examination is in the early diagnosis of tuberculosis. A lack of physical signs cannot rule out the disease, for in minimal tuberculosis a normal physical examination is usual.

In 156 patients no tubercle bacilli were recovered, in 3 the records contained no reports, the patients having left the sanatorium before examinations could be made, and in 41 tubercle bacilli were found. They were reported either in smears of the sputum or gastric-contents concentrates or in cultures or both. The demonstration of tubercle bacilli is,

of course, absolute proof of tuberculosis, but the presence of a negative sputum does not mean that tuberculosis is absent. This is particularly true in minimal cases, as illustrated by the small percentage in this series without positive sputums. A more liberal use of cultures and utilization of guinea-pig inoculations would, however, have yielded a higher percentage of positive reports.

There are several important facts relative to the finding of tubercle bacilli. The number of positive reports depends on the frequency and manner of examination. A single sputum, if negative, is of no value in the diagnosis of tuberculosis. The longer the time used for the collection of sputum, the higher is the incidence of a positive sputum. The use of sputum concentrates alone is not satisfactory; fasting gastric contents must also be examined. This is essential whenever sputum concentrates are negative or there is little or no expectoration. Especially in minimal cases, little sputum may be raised or, as often occurs, the sputum may be swallowed. Sputums or gastric contents that are negative on concentration should be cultured, for many will thus be found to be positive. Moreover, repeated examinations are necessary for proper conclusions about the presence of tubercle bacilli. Should the findings still be negative and tuberculosis be suspected, guinea-pig inoculations of the sputum and gastric contents are required. The ascending order of sensitivity of the methods used in finding tubercle bacilli is as follows: direct smear, 24-hour sputum concentrate, 72-hour sputum concentrate, fasting gastric-contents concentrate, culture of sputum or gastric contents and guinea-pig inoculations. The comparison of the sensitivity of these tests has been described.[8, 9] In the 41 cases in which tubercle bacilli were found, 10 (24 per cent) were positive with sputum or gastric cultures alone, 9 (22 per cent) with gastric concentrates alone, and 4 with gastric concentrates combined with sputum and gastric-contents cultures. In other words, in 56 per cent of the patients with tubercle bacilli, they were revealed because of the examination of gastric contents and the utilization of cultures. Decker, Ordway and Medlar[9] report that the repeated use of cultures and guinea-pig inoculations of sputum and gastric contents disclosed bacilli in 70 per cent of active minimal cases.

It is not meant by this discussion that it is necessary to wait for a positive sputum to make a diagnosis of minimal tuberculosis, since an early x-ray examination will indicate the disease. Needless to say, in a case with a purulent sputum persistently negative for tubercle bacilli, tuberculosis is undoubtedly not present.

Other laboratory findings were of little help in diagnosis. The white-cell count was of negligible value. On admission to the sanatorium 148 patients had a count under 10,000, 25 had counts between 10,000 and 15,000, and only 1 patient had a count of more than 15,000. In 26 cases no count was available.

The sedimentation rate, although more sensitive than the white-cell count, was not diagnostic or indicative of clinical activity. Of the 200 patients, 89 (45 per cent) had a sedimentation rate of less than 10 mm. in one hour (Cutler method), 42 patients had a rate of 11 to 15 mm., 29 had a rate between 16 and 20 mm., and 40 had a rate over 21 mm., with all but 2 of these under 30 mm. Thus, 111 (56 per cent) of the patients had a sedimentation rate of over 10 mm.

The tuberculin test was apparently not used in the diagnosis of any patient, but it is possible that in some cases the procedure was carried out without being recorded.

A positive tuberculin reaction means only that a tuberculous infection has taken place in the body, with hypersensitivity to tuberculoprotein present. It provides no definite answer about the type, extent or activity of the disease. A negative reaction may occur in the presence of tuberculosis under special circumstances — if the test is done shortly after infection has taken place and before the tissues have become hypersensitive, in terminal tuberculous disease, in patients with healed tuberculous lesions, in intercurrent acute infections and in aged and debilitated persons. The percentage of reactors is also qualified by the amount of tuberculin, for the larger the dose the greater the number of positive cases. A maximum final dose of 10 mg. is required for definite conclusions, since a negative reaction with so much tuberculin usually rules out tuberculosis. The test is especially significant in infants and young children, for if the reaction is positive, it constitutes strong evidence of active tuberculous disease. Rechecking a negative case periodically sometimes shows a positive reaction, thus indicating the occurrence of tuberculous infection. The important point is that the tuberculin test used alone for diagnostic purposes misses many minimal cases, particularly in adults, and that x-ray examination is essential to make the diagnosis.

Fluoroscopy was recorded in 8 cases (4 per cent). In 7 of these it served as a corroborative measure in suspected cases of tuberculosis, and in 1 case it formed a routine examination in an apparently healthy person. This figure is probably not a true indication of the use of the fluoroscope in diagnosis, since the patients no doubt were not specifically questioned about it.

There have been divergent opinions concerning the value of fluoroscopy as a method of early diagnosis. Garland[10] has emphasized the following limitations of this procedure. Its efficiency depends to a moderate degree on the technical factors, and to a high degree on the time taken by the examiner to get his eyes accommodated to the fluoroscope. There is much variation in the visual acuity of physicians. Fluoroscopy is much less accurate than

radiography, even when carefully done by trained persons. Published studies of fluoroscopy and roentgenographic checkups show that in the hands of experts errors vary from 13 to 35 per cent. The highest number of errors occur in minimal cases, especially those with apical or supraclavicular lesions. Fluoroscopy is especially difficult in obese, thick-chested patients. From the viewpoint of economy, it actually does not cost much less than x-ray examination if allowance is made for the physician's time required for eye adaptation, examination, discussion and a report of the case. Moreover, the cost of missed cases is greater than the cost of x-ray examination.

Bloch and Tucker[11] have affirmed the value of fluoroscopy. They found tuberculous lesions of the reinfection type in 4.1 per cent of 15,000 clinic patients. Of these cases, 1.4 per cent were clinically significant and 58.6 per cent were minimal. These authors stress the following points. The fluoroscope is easy and economical to operate and is extremely adaptable to the practicing physician's office. Its routine use would be a significant diagnostic contribution. It has additional value because an ordinary film may miss a lesion hidden by the scapula, clavicle, ribs or heart, whereas with the fluoroscope the patient can be rotated and the clavicles elevated or depressed.

Daniels[12] emphasizes that the physician in general practice can be an extremely valuable case-finding agent, and considers the large group of patients that come to physicians' offices a fertile field for mass surveys. He estimates that 30 per cent of the general population visit a physician at some time during the year. He reports a study wherein every patient entering his office, irrespective of the complaint, was fluoroscoped and x-ray photographs were taken of all chests with suspicious lesions. In 250 patients, 7 active cases of pulmonary tuberculosis were found. Of these patients, none had a history of contact, 4 had positive sputums, 6 had no physical findings, 3 had an increased sedimentation rate, and 5 had no history indicating pulmonary disease. Daniels admits the errors of fluoroscopy, and advocates having an x-ray miniature-film machine available in each locality for examining all patients visiting private physicians. Fluoroscopy is of definite value and is much better than a physical examination for the discovery of minimal cases, but it is imperfect and cannot be relied on solely for diagnosis. To be certain of the presence or absence of tuberculosis, it would be necessary to x-ray patients considered negative on fluoroscopy.

X-ray examination was the main method employed in diagnosis, having been used in 187 (94 per cent) of the 200 cases in this series prior to admission to the sanatorium. In 97 of these, the film was utilized in a corroborative manner to determine the presence of tuberculosis suspected because of other factors — symptoms, physical findings and so forth. In 90 cases, the examination was routine, consisting of a contact examination, survey, pre-employment investigation or a Selective Service checkup, and the diagnosis was made in persons apparently healthy.

The main problem in the control of tuberculosis is that of early diagnosis. In addition, treatment that is necessary should be provided without delay, either in the home, if the conditions there are suitable, or in an institution. The physician has the duty to educate the patient and his family in the infectiousness of the disease and to convince them of the value and necessity of immediate care. A patient may delay proper attention at home, postpone seeking admission to the sanatorium or continue to work after the diagnosis because necessary home adjustments, such as adequate living arrangements, placement of children, payment of rent, purchase of food and clothing, storage of furniture, contact examinations and family medical care, have not been made. To meet these situations requires the co-operation of public or voluntary social-service and welfare associations and of public-health nursing and medical groups. If such problems are taken care of, patients will be prompted to accept medical treatment as soon as the diagnosis has been made. The possibility of progression of the disease can then be avoided and the morbidity and mortality of advanced tuberculosis thereby avoided.

Comment

This study has shown that the most important and reliable instrument in the early diagnosis of minimal tuberculosis is x-ray examination. Certainly in over 99 per cent of minimal cases it can effect the diagnosis, often long before other clinical or laboratory findings have indicated the disease. No lung examination can be considered complete without an x-ray examination, and a patient should never be told that tuberculosis is absent unless one has been done. This procedure should be employed in every patient with any symptoms suggestive of tuberculosis, in any illness that tuberculosis may simulate and periodically in all contact cases. Minimal tuberculosis is a symptomatic disease, but to diagnose all persons, including those in an asymptomatic phase, routine mass x-ray surveys are necessary.

Ninety-one (46 per cent) of the patients in this series were diagnosed on a routine examination, — 90 by x-ray and 1 by fluoroscopy, — indicating the value of routine investigation. These were apparently healthy persons, unsuspected of having an illness, in whom the tuberculosis was accidentally discovered through x-ray examination. Without this fortuitous circumstance, many patients would no doubt have eventually been diagnosed as advanced cases. That routine x-ray surveys find active minimal lesions is an established fact. Of

these 91 patients, 55 had symptoms, 21 were shown to be positive for tubercle bacilli, and 38, including 14 with progression, showed x-ray changes.

The greatest advance in tuberculosis in recent years has been the lesson taught by the mass x-ray surveys. These surveys, applied to Selective Service applicants, to the industrial population, to those to be employed, to those admitted to general hospitals and so forth, have found evidences of clinically significant pulmonary tuberculosis of reinfection type in at least 1 per cent of the cases. Most important is the fact that 65 per cent of the cases have been diagnosed in a minimal stage. The statistics of the surveys indicate that there are many hundreds of thousands of unrecognized and unsuspected cases of tuberculosis in the country that can be detected by routine x-ray examinations. Moreover, rapid, simple, economical and diagnostically accurate methods have been developed, through the use of 14-by-17-inch paper films and 4-by-5-inch and 35-mm. — and recently 70-mm. — miniature films taken with photofluorographic technic. The routine use of x-ray examination is not an impractical ideal but an absolute necessity and a judicious economy. There is a much greater cost — to the patient, his family and the public — in a missed or delayed diagnosis of tuberculosis than in the expenditure for films that reveal no lesions. It is hoped that in the near future the means will be found to arrange for routine and periodic radiographic examinations of the entire population of the country. When this is done, a great step forward will have been taken in the early diagnosis and eradication of tuberculosis.

Summary and Conclusions

The advantages of the early diagnosis of minimal tuberculosis are enumerated.

To determine the factors that could aid early diagnosis, 200 cases with minimal lesions have been studied and the results reported.

Tuberculosis should be suspected at any age, in any occupation and with any type of social or economic background.

Positive contact is not a clear diagnostic indication and was present in only 34 per cent of the patients. Close observation is, however, necessary for contact cases, since the incidence of tuberculous infection among them is high.

Minimal tuberculosis is a symptomatic disease. Eighty-two per cent of these patients had symptoms. This is contrary to the oft-repeated statement that minimal tuberculosis is asymptomatic. The complaints usual in tuberculosis are either acute or not arresting. The public should be taught the importance of symptoms.

Minimal tuberculosis can masquerade as one of many other ordinary illnesses.

Physical findings are totally unreliable in diagnosis. Only 29 per cent of the patients in this series had rales.

Tubercle bacilli were found in 21 per cent of the cases. The percentage of positive reports depends on the type and number of examinations. Sputum smears alone are of very little value. Concentrates and cultures — and ideally guinea-pig inoculations — of sputum and fasting gastric contents are required for an adequate search for tubercle bacilli.

The white-cell count and sedimentation rate are of little diagnostic help.

The tuberculin test received almost no use in the diagnosis of these cases.

Fluoroscopy has its value but is an imperfect diagnostic method.

The vital role of the practicing physician as a tuberculosis case-finding agent has been indicated.

Prompt acceptance by patients of medical treatment is essential.

X-ray examination was used for diagnosis in 94 per cent of the cases before admission. This is the only reliable method for early diagnosis of minimal tuberculosis. Films should be taken in every case with symptoms suggestive of tuberculosis, in any illness that may be tuberculosis and periodically in all contact cases. Routine radiographic examinations are required for the entire population. The value of mass x-ray surveys has been demonstrated.

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PENTOTHAL SODIUM ANESTHESIA IN NEUROLOGIC SURGERY

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THE purpose of this article is to summarize our experience with Pentothal Sodium anesthesia in neurologic surgery. Since this kind of anesthesia was first used in this clinic for pneumoencephalography,[1] its use has been extended to include all neurosurgical procedures, regardless of length, in patients over ten years of age.[2] Although this report is based on a planned study of 101 unselected cases in which Pentothal Sodium was the anesthetic agent, we have employed it in several hundred additional major neurosurgical procedures. No death could be attributed to the anesthetic.

The types of operation are listed in Table 1, as well as the age incidence, the duration of the anesthesia and the dosages of Pentothal Sodium.

TABLE 1. *Summary of Data.*

FACTOR	TYPE OF OPERATION: SUPRATENTORIAL CRANIOTOMY	SUBOCCIPITAL CRANIOTOMY	SECTION OF FIFTH SENSORY ROOT	LUMBAR LAMINECTOMY FOR PROTRUDED INTERVERTEBRAL DISK	DORSAL SYMPATHECTOMY	LAMINECTOMY FOR CORD TUMOR OR CERVICAL DISK	MISCELLANEOUS*	TOTALS
	30 Cases	11 Cases	21 Cases	10 Cases	6 Cases	6 Cases	17 Cases	
Age incidence (years):								
11–20	2	1			1		2	6
21–30	5	3		1		1	4	14
31–40	9	2	2	2	4	2	6	27
41–50	5	3	3	5	1	2	4	23
51–60	8		7	2		1	1	19
61–70	1	2	6					9
71–80			3					3
Duration of anesthesia (hours):								
½ or less			7					7
½ –1	2	2	11	6			10	31
1 –1½	10	2	3	3	2	3	2	25
1½–2	7	2		1	3	2	3	18
2 –2½	5	3			1	1	2	12
2½–3	3	2						5
3 –3½	3							3
Dosage of 2.5% Pentothal Sodium solution (cubic centimeters):								
11–20			1					1
21–30	1		3				1	5
31–40	1		5	3			2	11
41–50	2	3	5	2	1		2	15
51–60	9	1	4	2	2		1	19
61–70	4	2	1			2	3	12
71–80	2	3	2		1	1	4	13
81–90	4			1	1	1	1	8
91–100	4	1		1				6
101–110	1	1			1	1		4
111–120						1	1	2
121–130	1						1	2
131–140	1						1	2
141–150				1				1

*Includes Naffziger operation for exophthalmos and tantalum cranioplasty, 3 cases each; cordotomy, section of superficial pitrosal nerve, subtemporal decompression, drainage of subdural hematoma and spinofacial anastomosis, 2 cases each; and section of eighth nerve, 1 case.

PREOPERATIVE PREPARATION

The night before operation a cleansing enema and 0.1 gm. of Seconal Sodium are given. The meal before operation is omitted and 0.1 gm. of Seconal Sodium is given two hours before operation. Forty-five minutes before operation adults receive 0.65 mg. of atropine and children a proportionately smaller dose. When the patient is extremely nervous or upset, morphine is given with the atropine.

After the patient has been placed in position and the operative site has been prepared, a suitable vein in the hand or forearm is selected for the intravenous administration of 5 per cent dextrose in saline solution. A three-way stopcock is interposed between the intravenous needle and the dextrose solution. Rubber tubing leads from the stopcock to a 20-cc. syringe containing the Pento-

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thal Sodium. For the last few years we have used nothing but a 2.5 per cent solution of this anesthetic agent.

After the patient has been anesthetized and draped, the line of the incision is thoroughly infiltrated with 1 per cent novocain solution and the operation is begun. A Geudel airway is usually placed in the patient's mouth for the administration of oxygen if necessary.

Operative Procedures and Results

Subtentorial Craniotomy

In our experience, Pentothal Sodium anesthesia has been superior to all others for craniotomies. Because the induction of anesthesia is brief, the patient is under an anesthetic for a shorter time than would be the case if inhalation anesthesia were used. Moreover, since with this anesthetic agent there is no increased intracranial pressure, bleeding is reduced. If used together with local anesthesia, the Pentothal Sodium anesthesia need not be deep and may be lightened after the scalp flap has been reflected. During closure of the scalp wound, the anesthetic may again be deepened if necessary. When the patient is returned to bed, his head is elevated 45 cm. to lower the venous pressure and reduce postoperative oozing. Postoperative vomiting and retching, which are frequent after inhalation anesthesia, cause severe fluctuation in intracranial pressure and increase the tendency to postoperative bleeding. After Pentothal Sodium anesthesia vomiting is an unusual occurrence.

In suspected brain tumors encephalography is frequently employed as a diagnostic procedure. The encephalogram is made in the operating room, and craniotomy is performed immediately after the roentgenograms have been made. This naturally increases the duration of the anesthesia. In 3 sample cases the relative quantities of 2.5 per cent Pentothal Sodium used for pneumoencephalography and craniotomy were as follows: Case 1, 33 and 65 cc.; Case 2, 25 and 90 cc.; and Case 3, 35 and 60 cc.

That 2 patients in this series of thirty craniotomies required larger than average quantities was attributed to their excessive use of alcohol and tobacco. This is usually true of this type of patient. There were no untoward reactions to the drug in any of these cases.

Suboccipital Craniotomy

There were 11 suboccipital craniotomies in this series. Pentothal Sodium anesthesia was found to have the same advantages for this type of operation as for supratentorial craniotomies. In addition, the patient is able to co-operate in securing the proper position for operation. This is of great importance because the patient is placed in the sitting position for this operation. In this series the only complication was respiratory obstruction in an extremely obese patient, which was relieved by changing the position of the head.

Other Operations

Section of the sensory root of the fifth nerve in the middle fossa was done in 21 cases. The patient is not anesthetized until after the skin is prepared and the field draped. As soon as the sensory root is sectioned, the anesthetic is discontinued, denervation making the closure painless. Because many of these patients are aged and are poor surgical risks, a short anesthetic is of distinct advantage. There were no untoward reactions in this group.

Lumbar laminectomy for protruded intervertebral disk was done in 10 cases. Pentothal Sodium anesthesia has the disadvantage in this procedure of allowing little muscular relaxation. Accordingly, during the last nine months the operation has been done under spinal anesthesia, supplemented with Pentothal Sodium if the patient is very apprehensive or if the spinal anesthetic wears off before the operation has been completed. There were no reactions in this group.

Dorsal sympathectomy was performed in 6 cases. All the operations were performed with the patient in the sitting position. There were no reactions to the anesthetic.

Laminectomy was performed in 6 cases, either for cord tumor or for protruding intervertebral cervical disk. The only untoward reaction was labored respiration, which was relieved by changing the patient's position.

A patient who suffered from a traumatic head wound had continuous generalized convulsive seizures until 5 cc. of a 2.5 per cent solution of Pentothal Sodium was given. A patient who was being trephined for subdural hematoma developed hiccough and laryngeal spasm, which lasted for ten minutes. When the anesthesia was gradually deepened, both reactions stopped, and the patient's condition remained good for the rest of the procedure.

Summary

Intravenous Pentothal Sodium anesthesia has been found to be extremely satisfactory and safe for neurologic surgery.

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CLINICAL NOTE

AGRANULOCYTOSIS FOLLOWING MAPHARSEN THERAPY

A Report of Two Cases Treated with Penicillin

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AGRANULOCYTOSIS following Mapharsen therapy for syphilis has been a serious but fortunately rare occurrence. The concurrent use of penicillin in the treatment of this complication and of syphilis has not been previously reported. Hence, the following case reports are of interest:

Case 1. A 21-year-old Negro soldier developed a penile ulcer on May 23, 1944. *Treponema pallidum* was demonstrated in a dark-field examination on May 27. The Kahn reaction was negative on May 30. The past history was negative except for gonorrhea in September, 1943, which had been successfully treated with sulfonamides.

Except for the presence of a penile ulcer in the coronal sulcus, the physical examination on admission was essentially negative. The weight was 145 pounds. The blood pressure was 100/60.

Intensive therapy was begun on May 30 and consisted of the daily intravenous injection of 0.066 gm. of Mapharsen and the intramuscular injection of 0.2 gm. of bismuth subsalicylate in oil every 3 days. A mild Herxheimer reaction followed the first injection of Mapharsen. Treatment was given for 8 days without other adverse reactions. On the 9th day, the patient manifested a moderately severe Milian reaction, and arsenotherapy was omitted for 5 days. Arsenotherapy was resumed on the 14th day, when 0.030 gm. of Mapharsen was given. This was followed by a rise in temperature to 104.0°F. On the following day the patient was feeling well, and 0.030 gm. of Mapharsen was given without any adverse reaction. For the next 6 days, full doses of Mapharsen were given, — 0.066 gm. a day, — without any apparent ill effect. On June 19, the 21st day, the white-cell count was 3200, with 2 per cent neutrophils. That evening the patient complained only of slight anorexia. The temperature rose to 100.6°F. and the pulse to 80. Thereafter, no more arsenic or bismuth was given. A quantitative blood Kahn reaction had been positive (10 units) on the 13th day. In 21 days of therapy the patient had received sixteen injections of Mapharsen, totaling 0.984 gm., and seven injections of bismuth subsalicylate, totaling 1.4 gm.

On the 22nd day, anorexia was more marked and the patient complained of pain and swelling in the region of the left submaxillary lymph nodes. On physical examination he did not appear to be in distress. There was moderate redness and puffiness of the gums, and slight injection of the pharynx. The left submaxillary lymph nodes were tender and slightly swollen. During that day the patient did not feel especially ill and had to be forced to remain in bed. The temperature fluctuated between 102 and 103°F. and the maximal pulse was 96. The white-cell count was 3200 with no granulocytes. Treatment on the 22nd and 23rd days consisted of four daily intramuscular injections of 100 mg. of B. A. L. in oil. On the 23rd day, the temperature remained elevated and reached a maximum of 103°F., the pulse rose to 112. By that time, the patient looked acutely ill and complained of weakness and nausea. The other signs and symptoms had progressively increased. The white-cell count was 4600, with 1 per cent nonsegmented neutrophils. On the 24th day, the weakness had increased and prostration was marked. It became increasingly difficult for the patient to open his mouth because of the pain and swelling of the left submaxillary lymph nodes. Examination of the mouth and pharynx was essentially the same as previously noted. Neither ulceration nor necrosis was seen. Treatment on that day consisted of the intravenous injection of 1500 cc. of 10 per cent glucose in saline solution, in addition to two injections of 100 mg. of B. A. L. in oil. The white-cell count was 3300, with no polymorphonuclear leukocytes. In the evening the prostration had increased and the patient appeared critically ill. The temperature was 103°F., and the pulse 112. Forty thousand units of penicillin was given intramuscularly, and repeated every 3 hours for 7½ days.

On the 25th day, there was a decrease in the pain and swelling of the left submaxillary lymph nodes, and prostration was less evident. The maximum temperature was 102°F. In addition to penicillin, the patient was given two intramuscular injections of 100 mg. of B. A. L. in oil and a transfusion of 500 cc. of citrated blood. The white-cell count prior to the transfusion was 3900, with 2 per cent segmented neutrophils and 2 per cent eosinophils. On the 26th day, improvement continued and the maximum temperature was 101.6°F. The white-cell count on the following day was 4000, with 3 per cent segmented neutrophils, 13 per cent nonsegmented neutrophils, 2 per cent eosinophils and 32 per cent juvenile forms. The highest temperature reached on the two succeeding days was 99.8°F., and the dramatic clinical improvement that followed paralleled the improvement in the blood picture.

After the 28th day, the patient was afebrile and asymptomatic except for slight, decreasing weakness. Penicillin therapy was completed on the 32nd day. By the 37th day the penile ulcer had healed, physical examination was essentially negative, and the Kahn reaction was negative. The patient was discharged to duty on the 39th day.

In the treatment of the agranulocytosis the patient had received 1200 mg. of B. A. L. in oil, 500 cc. of citrated blood and 2,400,000 units of penicillin (Table 1).

Table 1. *Hematologic Data for Case 1.*

Date	Red-Cell Count	Hemoglobin (Tallqvist)	White-Cell Count	Segmented Neutrophils	Nonsegmented Neutrophils	Lymphocytes	Monocytes	Eosinophils	Basophils	Myelocytes	Juvenile Cells
1944	*x10⁶*	%	*x10³*	%	%	%	%	%	%	%	%
5-30	4.2	85	9.9	—	—	—	—	—	—	—	—
6- 3	—	—	5.2	—	—	—	—	—	—	—	—
6- 8	4.4	75	11.5	75	15	10	0	0	0	0	0
6-13	—	—	9.6	71	5	17	3	4	0	0	0
6-15	—	95	—	—	—	—	—	—	—	—	—
6-19	—	—	3.2	2	0	54	25	15	4	0	0
6-20	3.7	75	3.2	0	0	41	55	0	4	0	0
6-21	4.4	80	4.6	0	1	49	50	0	0	0	0
6-22	3.3	75	3.3	0	0	49	49	2	0	0	0
6-23	4.3	70	3.9	2	0	51	45	2	0	0	0
6-24	4.2	90	4.0	3	13	46	4	2	0	0	32
6-26	4.3	90	10.1	23	11	30	12	9	6	6	3
6-27	4.6	90	13.7	58	11	21	2	1	0	5	2
7- 5	—	90	6.6	62	6	28	2	2	0	0	0

Case 2. A 23-year-old soldier developed a penile ulcer on August 4, 1944. *T. pallidum* was demonstrated in a dark-field examination on August 17. The qualitative Kahn reaction was positive on that day, and a quantitative test was positive (80 units) on August 22. The past history in-

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cluded an attack of hepatitis in 1942 following vaccination for yellow fever. The patient was a periodically heavy drinker of alcohol.

Except for the presence of two ulcers on the shaft of the penis and bilaterally enlarged inguinal lymph nodes, physical examination was essentially negative. The weight was 150 pounds. The blood pressure was 120/80.

Intensive therapy was begun on August 18, and consisted of daily intravenous injections of 0.065 gm. of Mapharsen and intramuscular injections of 0.2 gm. of bismuth subsalicylate in oil every 3 days. A mild Herxheimer reaction followed the first injection of Mapharsen. Treatment was given for 8 days without other adverse reactions. On the 9th day, the patient developed a moderately severe Milian reaction, and treatment with arsenic and bismuth was omitted and permanently discontinued. In eight days of intensive therapy the patient had received eight injections of Mapharsen, totaling 0.520 gm., and three injections of bismuth subsalicylate, totaling 0.6 gm.

Six days after the last injection of Mapharsen, the patient complained of anorexia and appeared slightly jaundiced. Urinalysis revealed a trace of bile. A diagnosis of toxic hepatitis due to Mapharsen was made. The appetite decreased as the jaundice grew more intense during the next several days. The liver became tender and palpable 1 fingerbreadth below the right costal margin in the midclavicular line. A low-grade fever continued until September 8, when the patient complained of a chill in addition to a sore throat and gums. The temperature rose to 104.6°F. Physical examination revealed, in addition to jaundice and an enlarged, tender liver, a moderately increased redness and swelling of the gums and pharynx. No areas of ulceration or necrosis were seen. The white-cell count was 3850, with 9 per cent segmented neutrophils and 3 per cent eosinophils. Penicillin therapy was immediately begun, and the patient was given 40,000 units intramuscularly every 3 hours for the next 7½ days. On September 9, he appeared better and the maximum temperature was 102.8°F. The white-cell count was 4650, with 7 per cent segmented neutrophils, 3 per cent eosinophils and a normal number of platelets.

On the following day improvement continued and the highest temperature was 100.5°F. The white-cell count was 3750, with 2 per cent segmented neutrophils, 1 per cent nonsegmented neutrophils and 4 per cent eosinophils. On September 11, improvement was still more evident and the temperature reached a maximum of 99.4°F. The white-cell count was 7400, with 11 per cent segmented neutrophils and 3 per cent eosinophils. The patient was afebrile on September 12 and remained so thereafter. He had no complaints after that date, and the liver was no longer palpable. The jaundice was still evident, although decreased in intensity. The white-cell count on September 12 was 8800, with 20 per cent segmented neutrophils and 4 per cent nonsegmented neutrophils.

The remainder of the convalescence was uneventful, and the blood picture rapidly returned to normal. Penicillin therapy was completed on September 15, at which time the quantitative blood Kahn reaction was positive (10 units). Clinical jaundice was not detectable after September 23. The penile ulcers had entirely healed and physical examination was essentially negative on October 3, when the patient was transferred to the rehabilitation section. He remained there for 1 week and was discharged to duty feeling entirely well. The patient received 2,400,000 units of penicillin in the management of the agranulocytosis and of the primary syphilis. No B. A. L. or blood transfusions were given (Table 2).

TABLE 2. *Hematologic and Other Data for Case 2.*

DATE	RED-CELL COUNT	HEMOGLOBIN (TALLQVIST)	WHITE-CELL COUNT	SEGMENTED NEUTROPHILS	NONSEGMENTED NEUTROPHILS	LYMPHOCYTES	MONOCYTES	EOSINOPHILS	BASOPHILS	MYELOCYTES	JUVENILE CELLS	PLATELETS	ICTERIC INDEX	SERUM BILIRUBIN
1944	*×10⁶*	%	*×10⁶*	%	%	%	%	%	%	%	%		*units*	*mg./100cc.*
8-18	—	—	8.3	63	2	31	4	0	0	0	0	—	—	—
8-22	4.7	100	—	—	—	—	—	—	—	—	—	—	—	—
8-25	5.1	100	6.6	67	0	27	0	6	0	0	0	—	—	—
8-29	4.8	90	6.6	66	11	22	1	0	0	0	0	—	—	—
9-1	—	—	—	—	—	—	—	—	—	—	—	—	45	4.3
9-3	—	—	—	—	—	—	—	—	—	—	—	—	54	6.3
9-8	4.6	95	3.8	9	0	77	9	3	2	0	0	—	32	—
9-9	4.3	85	4.6	7	0	58	32	3	0	0	0	Normal	28	—
9-10	4.0	80	3.7	2	1	66	27	4	0	0	0	—	—	—
9-11	3.8	80	7.4	11	0	72	14	3	0	0	0	—	—	—
9-12	3.9	90	8.8	21	4	46	29	0	0	0	0	—	—	—
9-13	4.0	85	11.0	16	12	22	16	8	0	4	21	—	—	—
9-14	4.5	—	10.4	11	13	32	12	9	0	3	19	—	—	—
9-15	4.5	90	11.2	37	11	24	13	9	0	0	6	—	—	—
9-16	4.3	90	11.4	46	6	29	9	6	0	0	4	—	15	1.4
9-17	4.6	80	12.8	57	4	23	9	5	0	0	2	—	—	—
9-18	4.3	85	10.0	58	2	33	2	5	0	0	0	—	—	—
9-19	—	90	8.0	61	1	28	3	5	2	0	0	—	—	—
9-24	—	—	5.4	55	0	34	4	6	1	0	0	—	—	—
9-25	—	—	—	—	—	—	—	—	—	—	—	—	13	—

Agranulocytosis has been recognized as a serious but rare complication following standard Mapharsen therapy for syphilis. Fatalities have been reported by a number of observers.[1–4] On the other hand, Stephenson, Chambers and Anderson[5, 6] reported a total of over two hundred and eighty-five thousand injections of Mapharsen with no fatalities due to agranulocytosis. Kasich[7] quotes Nelson's series of 4066 patients who had received over fifteen thousand injections of Mapharsen, in 2 of whom blood dyscrasias occurred. No cases of agranulocytosis were reported in a series of 4871 patients who had received intensive Mapharsen therapy.[8] Studies of the sternal bone marrow in 14 patients during intensive arsenotherapy revealed no significant effect on either the erythrocytic or the leukocytic series of cells.[9] Kasich[7] has reported 2 cases in which agranulocytosis followed standard Mapharsen therapy. The patients recovered following the use of sulfadiazine, but he does not mention any further therapy for syphilis. Kasich recommends frequent white-cell counts for all patients receiving Mapharsen. When intensive arsenotherapy was made the treatment of choice in this theater for patients with primary and secondary syphilis, the routine of making white-cell counts and differential counts at five-day intervals was adopted. Patients in this hospital received a diet low in fats but high in proteins and carbohydrates. No supplementary vitamins or drugs were used.

To promote recovery in patients who have developed agranulocytosis following the administra-

tion of toxic drugs, it is of paramount importance to discontinue immediately the use of the drug. Treatment with repeated blood transfusions and the administration of Pentnucleotide, adenine sulfate, liver extract, leukocyte cream and yellow-bone-marrow extract has not been uniformly successful. Many cases have ended fatally because of overwhelming septicemia, in the absence of circulating leukocytes. There have been some cases in which spontaneous recovery occurred. Thus, the control of septicemia is essential to the successful management of patients with agranulocytosis while awaiting the spontaneous or induced recovery of the hematopoietic function of the bone marrow. The sulfonamides have been successfully used in this respect,[7,10] even when the agranulocytosis has been associated with sulfonamide therapy.[11] Agranulocytosis following sulfonamide therapy has been frequently observed, but sulfadiazine is probably the least likely of the sulfonamides to cause serious toxic reactions. On the other hand, fatal granulocytopenia developing during the administration of sulfadiazine has been reported.[12–14] It has been suggested that penicillin might prove a better preparation for the management of severe infection in patients with agranulocytosis.[15]

Cases of agranulocytosis that developed following the use of sulfathiazole in which penicillin was successfully employed have recently been reported.[16, 17] In 2 cases, agranulocytosis complicating Mapharsen therapy for syphilis has been successfully treated with penicillin, but the amount administered was probably inadequate to eradicate the primary infection.[18]

The patient in Case 1 received a total of 1200 mg. of BAL in oil by intramuscular injections during the four days after the onset of agranulocytosis. This substance has a double pharmacologic action, first by converting arsenic within the tissues into a nontoxic compound, and second by increasing the elimination of arsenic in the urine. It has been demonstrated that this substance is not toxic in the dosage given and is of distinct value in the treatment of arsenical dermatitis. There was reason to believe that its administration might be of value in other arsenical reactions; hence it was used in Case 1. There was even greater indication for its use in Case 2, because of the coexisting toxic hepatitis, but it was not employed and recovery did not seem to be retarded as a result. But the first patient had received a total of 0.984 gm. of Mapharsen prior to the onset of agranulocytosis, whereas the second had received only 0.520 gm. No definite benefit is claimed for its use, however, since the patient receiving it showed neither clinical nor hematologic evidence of improvement until penicillin was given in addition.

The principal reason for using penicillin in these cases was on account of its demonstrated value in the treatment of syphilis. Moreover, there was less risk than in using sulfadiazine, which is capable of producing agranulocytosis. These patients were therefore given penicillin according to the routine that has since been adopted as the one of choice in all patients with primary or secondary syphilis. Sepsis was promptly controlled in both patients, and Case 1 demonstrated a reversal of serologic reaction from positive to negative at the conclusion of treatment. In Case 2, the titer of the Kahn reaction dropped from 80 to 10 units during treatment. It seems probable that this patient has attained seronegativity without further treatment, but this cannot be definitely stated because follow-up serologic reports are not yet available.

Summary

Two cases of acute agranulocytosis — one with coexisting toxic hepatitis — attributable to Mapharsen therapy for syphilis are reported. Penicillin was of distinct value, both in promoting recovery by combating sepsis and in the treatment of the underlying syphilis.

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SYMPOSIUM ON MEDICAL SOCIOLOGY

SOME CLINICAL ASPECTS OF SOCIAL MEDICINE*

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THE English language sometimes seems to obstruct the processes of thinking. Words may become impoverished, or they may become so weighted with a variety of meanings that when they reach the auditory equipment of a dozen persons they carry twelve different meanings and so cloud mutual understanding. "Social" is one of those words. It would have been helpful to me in tackling the subject of social medicine if I could have known beforehand what the term "social" as related to medicine means to each of you. One has only to refer to the dictionary to find the reason for some of the confusion in its use, for there are given therein at least eleven varieties and shades of meaning. One synonym is given as "convivial." You may judge whether that is pertinent to our discussion today.

For our purpose we might as well leave it that the term "social" is not definitive. Let us accept it as having to do with human interrelations, and pass on to my contention that the phrase "social medicine" is in a sense ambiguous. We may presume to use the phrase "scientific medicine" if we wish to specify one focus of attention within the broad term "medicine." But what about "social medicine," a common phrase these days, and now dignified by a highly significant development at Oxford in the establishment of a professorship of social medicine? In the *British Medical Journal* for November 20, 1943, Dr. John A. Ryle, who holds that chair, has given an excellent statement of his use of the term in his article, "Social Medicine: Its meaning and scope." I have reason to be deeply interested in his presentation, and I can accept the utility of the phrase at this stage in a rethinking of our concept of medicine.

But before I deal with my assignment of addressing you on the subject of social medicine, I wish to register my reasons for objection to the term as ambiguous. Let us accept the brief dictionary definition of medicine in its broad inclusive meaning as "the science and art of dealing with the prevention, cure and alleviation of disease to man." How, then, can medicine as such be other than social? How can it be divorced from its social, its human and community, significances and still be so defined? Even the research laboratory worker, with his attention focused on the study of the blood, serves the goal of relief of human ills. We may ask how an education in medicine can be justified if it fails to keep clear the ultimate humanitarian purposes of that education.

My desk at the hospital is so placed that I can readily see the ambulance rushing up to the Emergency Ward at any time of the day. It is a rule of the parking space that there must be no obstacle to the approach to the Emergency Ward entrance, where at any moment, day or night, medical care is immediately available for the patient in emergency need, no matter who he is. There is no moralistic question whether he is "worthy" of medical skill, no question of his economic status. He is a patient. That is enough. Other less important questions may have to be considered later, but the immediate urgent medical need is the clear focus of attention. And why do we so easily take this for granted and assume that there is nothing unique in it? Why all this meticulous care? It is characteristic of thousands of hospitals all over the land, and it is a high tribute to the medical profession that this is so.

Obvious and accepted facilities such as these are so characteristic of our social life that we accept them without due appreciation of their significance. There must be meaning in this devoted attention of doctors and nurses to saving human life. What is the reason? Surely not the mere preservation of biologic existence. How tragic it is when the highest medical and nursing skill leaves the patient nothing but breathing and heartbeat, with little capacity to play his part in his social relations. The essential purposes of physicians, surely the "flower of our civilization," as Stevenson termed them, rest in something more than the saving of life itself. Is it not a tacit recognition of the meaning of life? And the deepest meaning of life for us human beings is in our personal, social relations. Its meaning would be bare if we were stripped of our immediate family and those dearest to us, or if our work and play had no comradeship in friendships made more deeply significant through mutual experiences.

It is in terms of disturbances in these relations that patients bring to the hospital with their diseases that I wish to consider the practice of medicine as you are to see it in your hospital experience. The focus will be on the effects of illness on personal and community life and the more distinctly social aspects of medicine. This focus should not blind us to the broad significance of the hospital as bringing to bear on the patients' problems all the wealth

*This is the first of a series of nine lectures on medical sociology given weekly at Harvard Medical School during January, February and March, 1945; they were sponsored by the Department of Preventive Medicine and were primarily intended for third-year students. These articles will temporarily replace the reports "Medical Progress."

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of scientific medical knowledge organized to the purpose of making this knowledge and skill available to those patients who seek its beneficence.

The history of medicine is swift of change. Medical knowledge in its scientific aspects has expanded prodigiously through scientific research, to such an extent that those responsible for the curriculum have an impossible task of holding the essentials for medical education in its fullest sense within the nonelastic limits of time allotment. Choices must be made. At the moment it is the science of medicine that has won out. There is danger that medicine will be thought of as synonymous with the scientific aspects of medicine alone, and not be left free to its broader definition, with scientific medicine held in its true relation to the whole subject. Heroic efforts are being made by many of your clinical leaders to combat the crowding out of these social and humanitarian aspects of medicine. Dr. Ryle in his definition of social medicine ably analyzes the situation in medicine today. He contends that as clinical medicine resorts more and more to technical procedures there is less and less intimate understanding of the patient as a person. "Science without humanism," he writes, "may work with atoms but it will not work with men." He argues for recognition of the "interdependence of clinical, social and environmental study" of the patient for "the contribution which each can make to a better appreciation of etiology and prognosis and to a better organization" of medical care. He maintains:

> Social medicine means what it says. It embodies the idea of medicine applied to the service of man as *socius*, as fellow or comrade, with a view to a better understanding and more durable assistance of all his main and contributory troubles which are inimical to active health and not merely to removing or alleviating a present pathology. It embodies also the idea of medicine applied in the service of *societas*, or the community of men, with a view to lowering the incidence of all preventable disease and raising the general level of human fitness.

One of the correctives that has been resorted to in the Boston hospitals is the inclusion in the medical team of the medical social worker, who is charged with responsibility to bring to the clinician's attention information about the patient as a person in his social setting. Her purpose is to bring into clearer focus the meaning of illness to the patient — its effect on his usual way of life, his responsibilities and unfulfilled family obligations. It is out of such experience as a medical social worker that I speak to you today.

It is *not* the function of the medical social worker to take over all the social responsibility for care of the patient. The patient is the responsibility of the physician. The more aware the latter is of the significance of social factors in the patient's life as they may bear on his illness, the more readily will he make suitable use of the assistance of the social worker. He turns to her to elaborate the brief — too brief — social study of the patient and to assist him and the patient in working out a practical plan supportive of his medical-care plan.

What are some of the methods of securing pertinent social information? One principle we must accept is that of *individualizing* the patient. For the patient this is entirely acceptable. The patient feels unique: he *is* unique. We can lose ourselves in the group in some situations, — ball games or symphony concerts, — but in illness we are unique. The feeling of the patient about his illness — its meaning to him — is then of first importance.

The trained medical social worker disciplined by working closely with expert clinicians has special reason to appreciate some scientific procedures and principles. She is trained to case study in the area of the social life. She keeps in mind the common need for a normal, satisfactory way of life — namely, satisfying family relations, health, economic independence with good occupational adjustments, freedom in recreational activities and a reasonable degree of fulfillment of one's wishes and interests. The patient's so-called "social situation" prior to illness and as effected by illness is of concern to the medical social worker. She must determine what aspects of this situation may be significant to the physician. The gathering of such data from the patient, his family and others, the correlation of them to the clinical findings in conference with the physician, and planning with the patient and the physician to make available to the patient her experience and resourcefulness are her suitable functions.

I think of the Greek chef who was sent into the hospital ward for study of gastric ulcer. After a few days there, he insisted in broken English that he must go home. A period of three or four weeks of carefully regulated regimen under observation on the ward was advised. The reasons for his resistance to the medical plan were brought to light when his Greek physician and a Greek priest were brought into consultation with the physician in the wards. There was the patient's fear of operation, but chiefly there was his anxiety about the welfare of his wife and three children. After the ward physician's careful explanation of the reason for the hospital stay had been interpreted to him and his wife, and he had received reassurance that his job in a Greek restaurant would be held for him, he calmly accepted the medical plan.

History-taking in medical experience as practiced in our busy wards is almost entirely concerned with the restricted medical situation. The social history is meager and often inadequate. The patient, a highly valuable source of information, may not give the full story. I will give you a case in point that illustrates the nature of social evidence.

A girl of sixteen with ulcerative colitis was admitted to the ward for study and treatment. The original social data on the medical record under "Family History" stated that the mother had died

of possible heart trouble at thirty-two; that the father was living and had recently been discharged from the Army for "malaria"; that there were two brothers — one with possible kidney trouble; and that a sister had died of pneumonia at the age of five.

Under "Social History," it was recorded that after the death of the mother, the father found it impossible to work and to care for his daughter. The patient was placed in a boarding school, where she said she was quite happy. Following this, she lived with a foster family for five months and was quite dissatisfied, since these people were allegedly unkind to her. For the last six months she had lived with another foster family, in which she was contented and happy. At the time of the report she was attending junior high school, where she was in the tenth grade.

Social investigation through agencies that had been concerned with the family for many years — among them the Society for Prevention of Cruelty to Children — revealed the following facts. The mother deserted the family when the patient was ten years of age; the father, a seafaring man of low intelligence and uncertain responsibility, was unable to care for the children and gave them up to an institution for destitute children. The five-year-old sister was legally adopted; a favorite brother was later placed in a foster home and the patient lost track of him. In 1942, she was placed in a private semidisciplinary home for girls because of her behavior, and since 1943 she had been placed in two different foster homes in an attempt to meet her obvious need for a home and family life.

The patient's own first report of her history was readily understood to express her desire to create out of her fancy a satisfactory family background. It could have served no good purpose to confront this extremely sick girl with the social evidence at hand, but this evidence served well the clinician and psychiatrist, who through the data given by the patient and the facts secured by the medical social worker were able to estimate the psychic trauma of childhood for this sensitive girl. Just as the dread evidence of cancer is left with the surgeon to disclose in due time to the patient or her family, so the psychiatrist may guard social facts until, having gained the patient's full confidence, he deems it wise to face those facts with her and to help her in achieving a more realistic attitude toward life.

Social factors may be etiologically significant, especially in psychosomatic medicine. Surely there are causal factors in disease to be found in an unwholesome physical environment and in industrial hazards, as well as in lack of decent recreational facilities for youth while alluring evil influences flourish around them. Possibly the term "ecology" may serve the purpose better. I further suggest that diagnosis in its fullest meaning would be more significant if the pathologic entity were placed in the setting of the adverse social factors. The question is not only what kind of patient has the disease but what difficulties beset him in his social relations. Surely one could get a fairer estimate of prognosis if these factors were taken into account and the social worker could help plan social treatment with the patient, in close collaboration with the physician in his medical-treatment plan.

The importance of the patient's participation in treatment is becoming increasingly significant. The old days of pills and powders have passed. As scientific knowledge progresses, as specific dietaries are recognized as therapeutic and as chronic diseases such as arthritis call for a carefully regulated regimen of rest or specific exercises, the patient must share enhanced responsibility for successful treatment. Obstacles to treatment, whether in the patient's attitude or in his social situation, must be overcome.

Last winter a forty-two-year-old patient came to one of our hospitals from a nearby state for treatment of hypertension. He was found to be an excellent candidate for the Smithwick operation. His medical record contained these notes under "Social History": "Married and five children, living and well. Lost present job for medical reasons, couldn't keep up. Afraid of dizziness."

The patient was referred to the social worker, with a report that he had refused the proposed plan for medical care when it was explained that it would mean a few months at home with diet regimen for weight loss, followed by operation with a carefully guarded and lengthy convalescence. The patient had had an industrial accident in 1937 that resulted in amputation of his leg, but in spite of this handicap he had returned to his skilled work in an aircraft factory. His job as drill-press operator entailed lifting heavy steel bolts and feeding them into the machine. He remained sitting while the machine was in operation, but was forced to stand repeatedly while it was being supplied with bolts.

The patient's return to this job was obviously out of the question. Fortunately, he was a resident of a state in which the revised Physical Rehabilitation Act was in effective operation. The surgeon's plan was offered in definite form, and the way was cleared for it through social planning with the patient, the physical-restoration supervisor and the Welfare Department under provision for Social Security Aid to Dependent Children. Careful following of a diet regimen made possible the operative procedure, and after suitable convalescence a program for vocational training in office work was begun.

It is easy to accept the principle that the patient's personal and social situation should be taken into account in the treatment of his disease. It is not so simple to put this into practice in medical care in our busy, modern hospitals and clinics. Time limitation is a serious factor. Focus of attention on

pathology, with due consideration given to minutiae, is time-consuming and tends to limit the area of attention, to the exclusion of a breadth of view. In the light of the vast realm of medicine, we must accept the necessity for specialization, with its deepening and enrichening of knowledge and of skills for merciful purposes. Specialization in the social aspects of medicine has come as a logical development of clinical specialization and the necessary organization of services to large numbers of patients in the present-day institutional practice of medicine. Medical practice deprived of specialized knowledge and skills is superficial indeed. How are the high value of specialization and the delegation of responsibility to be applied to adequate care of the patient? Only, I believe, through mutual respect for specialized knowledge and skills and a clear purpose to strive for unity in diversity.

We are a long way from effective integration in the medical care of our patients. The modern scientifically oriented teaching hospital, for instance, presents a complicated array of specialties within clinical medicine, together with several professional groups to whom special services are delegated. When the purpose of care of the patient is the common bond within this group, there is an opportunity for a high level of democratic functioning. The physician, the nurse, the medical social worker, the dietitian, the occupational and physical therapists, the librarian and the clergyman must each understand the function of the others and recognize without professional jealousies that there are areas of mutual responsibility. I firmly believe that the physician, who must be the leader of the group, should carry the primary responsibility in as broad a scope as his capacity and time allow, and that he should be the one to co-ordinate the special services that complement and supplement his own care of the patient. Such integration of special skills is not as yet the usual practice in modern hospitals.

The humanities as related to scientific medicine call for intelligent study, as one notes the trends of hospitals toward assumption of broader community-health services. In this trend there is sore need for the leadership of well educated medical men, men to whom education has yielded its most precious gift, open-mindedness and flexibility of thought, with a sensitive awareness of social responsibilities.

CASE RECORDS OF THE MASSACHUSETTS GENERAL HOSPITAL

Weekly Clinicopathological Exercises

FOUNDED BY RICHARD C. CABOT

TRACY B. MALLORY, M.D., *Editor* BENJAMIN CASTLEMAN, M.D., *Associate Editor*

EDITH E. PARRIS, *Assistant Editor*

NORMAL LABORATORY VALUES

To provide ready reference to the normal values of laboratory procedures recorded in the "Case Records of the Massachusetts General Hospital" and to the methods used the following tabular summary has been prepared* and is substituted for one of the cases. A limited number of reprints will be available and will be forwarded, so long as the supply lasts, to readers of the *Journal* on request, provided that an addressed and stamped (1½ cents) return envelope (approximately 4 by 9 inches) is enclosed. All requests should be addressed to Miss Edith E. Parris, Massachusetts General Hospital, Boston 14.

* * *

Blood, Plasma or Serum Values

DETERMINATION	MATERIAL ANALYZED	MINIMUM QUANTITY REQUIRED	NORMAL VALUE	METHOD
		cc.		
Amino acids (CO_2 of carboxyl carbon)	Plasma	2	3.4–5.5 mg. per 100 cc.	Hamilton and Van Slyke: *J. Biol. Chem.* **150**:231, 1943
Amylase	Serum	2	15–35 units per 100 cc.	Adapted from Somogyi: *J. Biol. Chem.* **125**:399, 1938.
Ascorbic acid (vitamin C)	Plasma	0.5	0.4–1.0 mg. per 100 cc. (fasting)	Butler, Cushman and MacLachlan: *J. Biol. Chem.* **150**:453, 1943
Ascorbic acid	White cells	8 (whole blood)	25–40 mg. per 100 cc.	*Ibid.*
Bilirubin (van den Bergh test)	Serum	2	Direct, 0.4 mg. per 100 cc.; indirect (total) 0.7 mg. per 100 cc.	Malloy and Evelyn: *J. Biol. Chem.* **119**: 481, 1937
Calcium	Serum	2	9.0–10.5 mg. per 100 cc.†	Fiske and Logan: *J. Biol. Chem.* **93**:211, 1931; Folin: *Lab. Manual Biol. Chem.*, 5th ed., p. 351.
Carbon dioxide (content)	Serum	0.5	26–28 meq. per liter‡	Van Slyke and Neill: *J. Biol. Chem.* **61**: 523, 1924; Peters and Van Slyke: *Quant. Clin. Chem.*, Vol. II (Methods), p. 283.
Carotenoids: Total	Serum	2	100–300 int. units per 100 cc.	Josephs: *Bull. Johns Hopkins Hosp.* **65**:112, 1939 (modified for photocolorimeter and calibrated with haliver oil of specified vitamin A content)
Vitamin A	Serum	2	40–100 int. units per 100 cc.	
Chloride	Serum	0.5	100–106 meq. per liter	Wilson and Ball: *J. Biol. Chem.* **79**:221, 1928
Cholesterol	Serum	0.5	150–230 mg. per 100 cc.	Bloor: *J. Biol. Chem.* **24**:227, 1916

*The chemical data were prepared by G. Margaret Rourke, chief of the Chemical Laboratory, Elsie A. MacLachlan, consultant, and Allan M. Butler, staff physician in charge of the Chemical Laboratory.

†Since the total serum calcium concentration is dependent on the serum protein concentration, evaluation of the significance of a serum calcium value requires a serum protein determination.

‡Values in infants and children may be from 20–26 meq. per liter. Milliequivalents per liter equal volumes per cent divided by 2.2. The milliequivalent total CO_2 minus 1.3 is usually about equal to the HCO_3 expressed as milliequivalents per liter. For reason for using milliequivalents as units of concentration here and for other electrolytes see Butler and Talbot: *New Eng. J. Med.* **231**:20, 1944.

Determination	Material Analyzed	Minimum Quantity Required	Normal Value	Method
		cc.		
Cholesterol esters	Serum	0.5	65 per cent of total cholesterol	Bloor and Knudson: *J. Biol. Chem.* **27**: 107, 1916
Glucose	Blood	0.1	70–100 mg. per 100 cc. (fasting)	Folin: *Lab. Manual Biol. Chem.*, 5th ed., p. 307; Folin: *New Eng. J. Med.* **206**: 727, 1932.
Hemoglobin	Blood	0.05	14–16 gm. per 100 cc.	Evelyn: *J. Biol. Chem.* **115**:63, 1936
Iodine, protein-bound (thyroid hormone)	Serum	4	4–8 microgm. per 100 cc.	Talbot, Butler, Saltzman and Rodriguez: *J. Biol. Chem.* **153**:479, 1944
Magnesium	Serum	2	1–2 meq. per liter	Briggs: *J. Biol. Chem.* **59**:255, 1924
Nonprotein nitrogen	Serum	0.5	15–35 mg. per 100 cc.	Folin: *Lab. Manual Biol. Chem.*, 5th ed., p. 265
Oxygen:				
Capacity	Blood	3	19–22 vol. per cent	Van Slyke and Neill: *J. Biol. Chem.* **61**: 523, 1924; Peters and Van Slyke: *Quant. Clin. Chem.*, Vol. II (Methods), p. 321.
Arterial content	Blood	3	18–21 vol. per cent	*Ibid.*
Arterial percentage saturation	—		94–96 per cent	(Arterial content x 100) ÷ capacity
Venous content	Blood	3	10–16 vol. per cent	*Ibid.*
Venous percentage saturation	—		60–85 per cent	(Venous content x 100) ÷ capacity
pH(reaction)	Serum	0.2	7.35–7.45	Hastings and Sendroy: *J. Biol. Chem.* **61**: 695, 1924; Peters and Van Slyke: *Quant. Clin. Chem.*, Vol. II (Methods), p. 796.
Phosphatase, acid	Serum	1	0.5–2.0 units per 100 cc.	Gutman and Gutman: *J. Biol. Chem.* **136**:201, 1940
Phosphatase, alkaline	Serum	0.5	2.0–4.5 units per 100 cc.†	Bodansky: *J. Biol. Chem.* **101**:93, 1933 (using the method for determining inorganic phosphorus)
Phosphorus, inorganic	Serum	0.2	3.0–4.5 mg. per 100 cc.*	Fiske and Subbarow: *J. Biol. Chem.* **66**: 375, 1925; Folin: *Lab. Manual Biol. Chem.*, 5th ed., p. 341 (modified for photocolorimeter).
Potassium	Serum	3–4	3.5–5.0 meq. per liter	Fiske and Litarczek in Folin: *Lab. Manual Biol. Chem.*, 5th ed., p. 353.
Protein:				
Total	Serum	0.5(macro) 0.05(micro)	6.5–8.0 gm. per 100 cc.	Macro: Peters and Van Slyke: *Quant. Clin. Chem.*, Vol. II (Methods), p. 691. Micro: Lowry and Hastings: *J. Biol. Chem.* **143**:257, 1942.
Albumin	Serum	0.5	4.5–5.5 gm. per 100 cc.	*Ibid.*
Globulin	Serum	0.5	1.5–3.0 gm. per 100 cc.	*Ibid.*
Prothrombin clotting time	Plasma	0.3	By control	Quick: *J. A. M. A.* **110**:1658, 1938
Pyruvic acid	Blood	2	0.7–1.2 mg. per 100 cc. (fasting)	Friedemann and Haugen: *J. Biol. Chem.* **147**:415, 1943; Bueding and Wortis. *Ibid.* **133**:585, 1940.
Sodium	Serum	0.5	136–145 meq. per liter	Butler and Tuthill: *J. Biol. Chem.* **93**: 171, 1931
Urea nitrogen	Serum	1	10–28 mg. per 100 cc.	Van Slyke: *J. Biol. Chem.* **73**:695, 1927; Peters and Van Slyke: *Quant. Clin. Chem.*, Vol. II (Methods), p. 372.
Uric acid	Serum	1	3–5 mg. per 100 cc.	Folin: *J. Biol. Chem.* **101**:111, 1933

*In the newborn infant values may be as high as 6 mg. per 100 cc., which then diminish during the first year; in childhood they approach the normal adult average value of 3.5 mg.

†The value parallels the rate of growth, diminishing from approximately 14 units per 100 cc. in infancy to 5 units in adolescence and thereafter being maintained at approximately 3.5 units.

Urine Values

Determination	Minimum Quantity Required	Normal Value	Method
	cc.		
Albumin (quantitative)	10	0	Folin:*Lab. Manual Biol. Chem.*, 5th ed., p. 225
Creatine	24-hr. sample	Less than 100 mg. per 24 hr.*	Folin:*Lab. Manual Biol. Chem.*, 5th ed., p. 163
Creatinine	24-hr. sample	15–25 mg. per kg.†	*Ibid.*, p. 159 (modified for photocolorimeter)
Diastase	2	Dilution of 1:4 to 1:16	Stitt:*Pract. Bact. Haem. & Parasitol.*, 9th ed., p. 731
Follicle stimulating hormone	24-hr. sample	Before puberty, less than 6.5 mouse units per 24 hr.; after puberty 6.5–52 mouse units per 24 hr.; after menopause 104–600 mouse units per 24 hr.	Klinefelter, Albright and Griswold: *J. Clin. Endocrinol.* **3**:529, 1943
Sugar:			
Total (quantitative)	5	0	Benedict:*J. A. M. A.* **57**:1193, 1911
Total (roughly quantitative)	0.5	0	Somogyi:*J. Lab. Clin. Med.* **26**:1220, 1941
Fermentable	1	0	Hawk and Bergheim:*Pract. Physiol. Chem.*, 10th ed., p. 750
Fructose	1	0	*Ibid.*, p. 772
Galactose or lactose	6	0	(Total sugar x1.24) minus fermentable sugar
Osazone, differentiation of	5	0	*Ibid.*, p. 50
Urobilinogen	10	Dilution of 1:4 to 1:30	Wallace and Diamond:*Arch. Int. Med.* **35**:698, 1925
17-ketosteroids	12-hr. sample	Under 8 yr., 0–2 mg. per 24 hr.; adolescents, 2–20 mg. per 24 hr.; males, 8–20 mg. per 24 hr.; females, 5–14 mg. per 24 hr.	Talbot, Butler, MacLachlan and Jones: *J. Biol. Chem.* **136**:365, 1940; Fraser, Forbes, Albright, Sulkowitch and Reifenstein:*J. Clin. Endocrinol.* **1**: 234, 1941.

*Per kilogram of body weight, the excretion is higher in women and children than in men, and still higher in infants.

†The value depends on the ratio of muscle to fat in the body mass of the patient. The higher the ratio the greater the creatinine excretion per kilogram of total body weight. Because this ratio is low in infants, the excretion per kilogram is low.

Liver-Function Tests

Determination	Amount Administered	Material Analyzed	Minimum Quantity Required	Normal Value	Method
			cc.		
Bromsulfalein*	2 mg. per kg. intravenously	Serum (30 min. after injection)	2	Less than 5 per cent retention	Rosenthal and White:*J. A. M. A.* **84**:1112, 1925; Peters and Van Slyke:*Quant. Clin. Chem.*, Vol. II (Methods), p. 910.
	5 mg. per kg. intravenously	Serum (45 min. after injection)	2	Less than 5 per cent retention	*Ibid.* (modified, *i.e.*, result÷2.5)
Cephalin flocculation	0	Serum	0.2	Up to ++ in 48 hr.	Hanger:*J. Clin. Investigation* **18**: 261, 1939

Determination	Amount Administered	Material Analyzed	Minimum Quantity Required	Normal Value	Method
			cc.		
Galactose tolerance	0.5 gm. per kg. intravenously	Blood	1	Less than 5 mg. at 75 min.	Basset, Althausen and Coltrin: *Am. J. Digest. Dis. & Nutrition* **8**:432, 1941
Hippuric acid	1.77 gm. sodium benzoate intravenously	Urine	1-hr. sample	Greater than 1 gm.	Quick, Ottenstein and Weltchek: *Proc. Soc. Exper. Biol. & Med.* **38**: 77, 1938; Moser, Rosenak and Hasterlik: *Am. J. Digest. Dis. & Nutrition* **9**:183, 1942.

*The 2-mg. method is used in patients with slight jaundice, and the 5-mg. method in patients without jaundice; the method is valueless in patients with obvious jaundice.

Renal-Function Tests

Determination	Amount Administered	Material Analyzed	Minimum Quantity Required	Normal Value	Method
			cc.		
Phenolsulfonphthalein	1 cc. intravenously	Urine	Total output	25 per cent or more in first 15 min.; 40 per cent or more in 30 min.; 55 per cent or more in 2 hr.	Chapman: *New Eng. J. Med.* **214**: 16, 1936
Urea clearance	0	Blood and urine	Blood, 1 cc.; urine, two 1-hr. samples.	75 to 125 per cent of normal	Peters and Van Slyke: *Quant. Clin. Chem.*, Vol. II (Methods), p. 564

Hematologic Values

Determination	Minimum Quantity Required	Normal Value	Method
	cc.		
Bleeding time	—	Below 4½ min.	Lee and White in Todd and Sanford: *Clin. Diag. by Lab. Methods*, 10th ed., p. 199
Clotting time	10	Below 20 min.	Duke: *J. A. M. A.* **55**:1185, 1910
Sedimentation rate (two methods)	4	Less than 0.35 mm. per min.	Rourke and Ernstene: *J. Clin. Investigation* **8**:545, 1930
	4	Less than 15 mm. per hr.	Modification* of Wintrobe and Landsberg: *Am. J. M. Sc.* **189**:102, 1935
Hematocrit (percentage volume of packed red cells)	2	Male, 40–54 per cent; female, 37–47 per cent.	*Ibid.*
Hemoglobin	0.05	14–16 gm. per 100 cc.	Evelyn: *J. Biol. Chem.* **115**:63, 1936
Mean corpuscular volume	—	80–94 cu. microns	(Hematocrit x10)÷red cells (in millions)
Mean corpuscular hemoglobin	—	27–32 micromicrogm.	(Gm. of hemoglobin x 10)÷red cells (in millions)
Mean corpuscular hemoglobin concentration	—	33–38 per cent	(Gm. of hemoglobin x 100)÷hematocrit

*Internal diameter of tube should be 4 mm. instead of 2.5 mm.

Spinal-Fluid Values

Determination	Minimum Quantity Required	Normal Value	Method
	cc.		
Initial pressure	—	70–180 mm. of water	
Cell count	0.2	0–5 mononuclear cells (lymphocytes)	
Chloride	2	120–130 meq. per liter	Same as that for serum (see above)
Protein	0.6	15–45 mg. per 100 cc.	Ayer, Dailey and Fremont-Smith: *Arch. Neurol. & Psychiat.* **26**:1038, 1931
Glucose	1	50–75 mg. per 100 cc.	Same as that for blood (see above)
Colloidal gold	0.1	0000000000	Wuth and Faupel: *Bull. Johns Hopkins Hosp.* **40**:297, 1927

Miscellaneous Values

Determination	Material Analyzed	Minimum Quantity Required	Normal Value	Method
		cc.		
Stool fat		Representative sample	Less than 30 per cent dry wt.	Tidwell and Holt: *J. Biol. Chem.* **112**: 605, 1936
Calculi		Representative sample		McIntosh and Salter: *J. Clin. Investigation* **21**:751, 1942
Congo red test	Serum	2	More than 60 per cent retention in serum after 1 hr.	Bennhold: *Deutsches Arch. f. klin. Med.* **142**:32, 1923

Case 32011

Presentation of Case

A seventy-four-year-old man entered the hospital complaining of jaundice.

Ten years before admission the patient underwent a cholecystectomy. Gallstones were found. During the years after the operation he had several attacks of jaundice, each lasting a few days. Seven months before admission he began to have increasingly severe attacks of epigastric pain, radiating to the chest and to the right costovertebral angle. He also noticed nausea and frequent gaseous eructations. He had lost considerable weight. Ten days before admission jaundice and itching appeared and rapidly increased in severity.

On physical examination the patient was emaciated, weak and deeply jaundiced. The heart was slightly enlarged to the left. A systolic murmur and a diastolic murmur were heard at the apex. The right chest was more prominent than the left, which showed lateral flattening. Rales were heard at both bases. The abdomen was flat and soft. The liver was tender and enlarged, extending four fingerbreadths below the costal margin. An upper-right-rectus scar and a herniorrhaphy scar were present. The reflexes were normal.

The temperature was 98°F., the pulse 75, and the respirations 20. The blood pressure was 150 systolic, 85 diastolic.

The urine was normal except for a ++++ test for bile. The red-cell count was 3,820,000, with a hemoglobin of 12.8 gm. The white-cell count was 7200. The serum bilirubin was 28.5 mg. per 100 cc. direct, and 30.3 mg. indirect. The prothrombin time was 24 seconds (normal, 18 seconds). The bleeding time was 2½ minutes, and the clotting time 4 minutes. Clot retraction was normal. The total serum protein was 6.5 gm. per 100 cc., with 4.3 gm. of albumin and 2.2 gm. of globulin. The nonprotein nitrogen was 19 mg. per 100 cc., and the chloride 95 milliequiv. per liter. A stool specimen was light brown and guaiac positive; it was slightly positive for bile.

An x-ray film of the chest taken on the first hospital day showed an area of hazy increased density

beneath the left fourth, fifth, and sixth ribs anteriorly. This portion of the lung fields, which was near the lateral chest wall, was somewhat obscured by the closely grouped overlying ribs. The remainder of the lung fields did not appear remarkable. The heart was not enlarged but was rounded in the region of the left ventricle. The aorta was tortuous. Another film taken two days later again showed a somewhat triangular-shaped area of increased density against the posterolateral chest wall on the left. A linear area of increased density was present in the left costophrenic angle posterolaterally. There was a hazy area of increased density just to the right of the heart, apparently in the middle lobe.

Soon after admission the patient became markedly orthopneic. After vomiting bloody, bile-tinged fluid, he expired on the fourth hospital day.

Differential Diagnosis

Dr. Francis D. Moore: This is the story of a man who had had a gall-bladder operation many years before entry and subsequently had a train of events that might or might not have been related to the cholecystectomy. He finally died of what appears, at least on the surface, to have been a third disease.

The history is of the greatest importance in trying to decide the cause of his jaundice. There are a few things not mentioned in the record that would have been a great help in that regard. We read that during the years after the cholecystectomy he had had several attacks of jaundice, each lasting a few days. We should like to know whether there was pain with the jaundice. If a patient has had a cholecystectomy and then postoperatively has bouts of pain and jaundice that pass off spontaneously, one is justified in thinking that a few common-duct stones were left in the duct, which he is passing off one after another, or possibly that he is suffering from that mysterious entity known as "biliary dyskinesia." If the attacks were painless, they were possibly attacks of cholangitis, in which case one would expect fever and possibly chills.

We should also like to know whether there was a long free interval between these postoperative attacks and the train of events that later led up to his last admission. In other words, was there a period of several years when he was free from pain and jaundice? This is important as it would make one feel reasonably confident that retained common-duct stones were not a factor in his fatal illness.

Dr. Ronald C. Sniffen: I might say in partial answer that Dr. E. P. Hayden saw the patient about four months before the jaundice developed. At that time the patient complained of vague abdominal symptoms. A complete gastrointestinal series was done, but nothing was found.

Dr. Moore: That tends to make persistent common-duct stones following cholecystectomy less likely.

In his final illness he had a rapid crescendo of pain and jaundice and had itching, a common symptom in extrahepatic biliary obstruction. From that point on. the record tells us of the physical findings and laboratory data in a man with noninflammatory extrahepatic biliary obstruction, which was incomplete.

My reasons for making these statements are that he had a normal white-cell count and temperature, and it is therefore reasonable to assume that he did not have a primary inflammatory process. The obstruction seems to have been extrahepatic rather than parenchymatous partly because of the high ratio of the direct to the indirect bilirubin, almost 90 per cent, which is quite characteristic of extrahepatic biliary obstruction, and also because with this high total bilirubin there was little evidence of liver-cell damage. The prothrombin time was slightly elevated, it is true, but the albumin-globulin ratio was normal. In other words, the degree of obstruction seems to have been out of proportion to the liver damage.

I do not believe that the obstruction was complete because the patient had bile in his stools; they were brown and showed a chemical test for bile, and just before he died he vomited bile-stained material, which also tells us that the pylorus was patent. We can therefore say that he had incomplete noninflammatory extrahepatic biliary obstruction and, in addition, bleeding into the gastrointestinal tract, since he passed guaiac-positive stools and vomited some blood just before he died. Incomplete extrahepatic obstruction and bleeding into the gastrointestinal tract should be absolutely diagnostic of carcinoma at the ampulla of Vater, but before we discuss what is wrong with that diagnosis, we might see the x-ray films of the chest.

Dr. Clayton H. Hale: The films taken at the first examination demonstrate a somewhat triangular shadow of increased density at the level of the anterior portions of the fourth, fifth and sixth ribs. In the lateral projection, this area of increased density is seen to lie posteriorly and there is a slight suggestion of rarefaction in the central portion of the mass. There is also evidence of a healed infarct in the left costophrenic sinus. The cardiac and vascular shadows and the remainder of the lung fields are not remarkable. No significant change is demonstrated on the films taken two days later. From the x-ray finding one must give first consideration to multiple infarcts but the possibility that the large area of density is due to metastatic cancer must be given serious thought.

Dr. Moore: How often do you see areas of diminished density in uninfected pulmonary infarcts?

Dr. Hale: They are unusual. The possibility of this being a septic infarct must be considered if there is actual rarefaction in the area. The possibility of an abscess, although it is not at all typical,

cannot be ruled out. A malignant neoplasm, either primary or metastatic, must also be kept in mind.

DR. MOORE: How often do you see areas of diminished density in metastatic cancer?

DR. HALE: We do not see them often.

DR. MOORE: The x-ray films are confusing. From the written record I thought that the x-ray description was characteristic of pulmonary infarction, and the clinical course suggests two or three warning attacks, with a subsequent massive infarct and death. The area that Dr. Hale says is suggestive of septic infarct and abscess is disturbing. It is difficult to believe that there was pulmonary suppuration in a man who had a normal temperature and white-cell count. I shall have to reserve judgment on the pulmonary lesion and say that it was probably due to metastatic cancer.

What was the fundamental disease in the abdomen? I have said that many of the characteristic findings were those of carcinoma in the region of the ampulla of Vater, but there are some things wrong with that diagnosis. In the first place, the patient had quite a lot of pain. Carcinoma in the lower portion of the common duct and head of the pancreas is usually accompanied by painless jaundice. There is one explanation, however, for pain in this man. With obstruction to the common duct, the gall bladder is ordinarily greatly distended, and part of the clinical picture of carcinoma of the head of the pancreas is a big gall bladder, which serves partly to decompress the biliary tree. This man did not have a gall bladder to distend. Possibly this absence of a gall bladder accounts for the pain. It is also interesting that we have seen several patients in the last two years who had had previous gallstones and in whom the gall bladder had been removed, and who several years later developed carcinoma lower down in the biliary tree. It makes one wonder if cholelithiasis is part of the background of biliary-tract carcinoma in general, as well as of cholecystic carcinoma itself.

How about common-duct stone? I have already mentioned that it would be unusual for him to have had a long period free of pain if he had had a common-duct stone. It is rare for a person to walk around with common-duct stones for ten years, because they are intermittently extremely painful and should have caused trouble much sooner. If we knew that the gall bladder contained large gallstones and that the cystic duct was normal at the first operation, I should be willing to rule it out completely.

How about other tumors? A tumor higher in the biliary tree, away from the ampulla of Vater, can bleed down the duct and into the gastrointestinal tract, but that is very rare. Carcinoma of the duodenum is a possibility, but it, too, is extremely rare. What about carcinoma of the pancreas? He certainly could have had a carcinoma of the pancreas that obstructed the duct, but because of the bleeding, I think that this was a lesion directly continuous with the lumen of the gastrointestinal tract. I have already discussed intrahepatic jaundice. Possibly his bleeding was from varices. How about carcinoma of some other organ? Dr. Linton* has described several cases of carcinoma of the colon and duodenum in which continuity between the duodenum and the transverse colon had been established, with a duodenocolic fistula and jaundice. There was no barium enema on this man; such a diagnosis, however, is only a remote possibility.

As I have said before, I think that he had pulmonary infarcts, but it is too bad that we have no mention of what the lungs showed on physical examination. It might help us to decide on the pulmonary lesion.

My diagnosis, therefore, is carcinoma in the region of the ampulla of Vater. I rule out a common-duct stone. I believe that the cause of death was a massive pulmonary infarct and that the previous pulmonary lesion was an infarct. But the latter, as Dr. Hale has pointed out, might have been a metastatic lesion or some form of abscess.

CLINICAL DIAGNOSIS

Carcinoma of pancreas.

DR. MOORE'S DIAGNOSES

Carcinoma at the ampulla of Vater, with direct invasion of the duodenum.
Pulmonary infarction?
Pulmonary metastases?

ANATOMICAL DIAGNOSES

Papillary adenocarcinoma of head of pancreas, with metastases to common bile duct and lung.
Early obstructive cirrhosis of liver.
Jaundice.
Bronchopneumonia.

PATHOLOGICAL DISCUSSION

DR. SNIFFEN: At autopsy the patient was deeply jaundiced. The liver was dark green and enlarged, weighing 2050 gm. The sections microscopically suggested an early obstructive cirrhosis. The common bile duct was dilated down to the ampulla, which admitted a fine probe. The head of the pancreas seemed a little larger than normal, but the body and tail were atrophic and fibrous. Sections taken through the head of the organ showed microscopically a papillary adenocarcinoma, whereas the parenchyma of the body and tail had undergone atrophy, leaving the islets embedded in dense

*Linton, R. R. Two stage operation for carcinoma of transverse colon producing duodenocolic fistula: report of two cases. *Arch. Surg.* 48: 197-207 1944.

fibrous tissue. There were metastases to the regional lymph nodes, and the wall of the common bile duct contained a 3-mm. nodule of metastatic tumor, which had not eroded the mucosa. I wonder if this small nodule could have caused the pain.

The lungs showed a bilateral bronchopneumonia concentrated in the lower lobes. The density mentioned in the x-ray report was due, I imagine, to a 4-cm. area of metastatic cancer in the left lower lobe.

DR. MOORE: Perhaps his death was not so sudden as the record indicates. Was the cause of death bronchopneumonia?

DR. SNIFFEN: I do not know why he died suddenly.

As incidental findings, the prostate was hyperplastic, with a large median bar, and one section contained a minute focus of primary carcinoma. The large median lobe had led to hypertrophy of the bladder, cystitis, a left hydroureter and ureteritis, and left acute and chronic pyelonephritis.

DR. MOORE: You found no cause for the bleeding?

DR. SNIFFEN: No; the duodenal mucosa was intact.

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PEPPER BILL

Appearing elsewhere in this issue of the *Journal* is an informational report by the Subcommittee on Medical Economics of the Postwar Planning Committee that deserves careful reading by every member of the Massachusetts Medical Society.

Last fall this subcommittee was faced with the responsibility of making recommendations concerning the Wagner–Murray–Dingell Bill (S. 1050) and the Pepper Bill (S. 1318). Since it was wisely decided that such action was impossible in the absence of clearly defined basic principles governing medical-care programs, the subcommittee proceeded to create a set of eight principles that appeared to be essential to any medical-care plan in a free society. Then, on the basis of these principles and of facts more generally regarded as necessary in medical care, the Pepper Bill was carefully studied.

As indicated in its report, the subcommittee favors the objectives stated in the introduction to the bill but it also believes, for various reasons, that the bill does not represent the best form of legislation to accomplish what is intended.

The report of the subcommittee has been accepted, with minor modification, by the Postwar Planning Committee, the Committee on Legislation and the Committee on Public Relations, and it will be presented to the Council of the Massachusetts Medical Society for acceptance as a joint recommendation.

All this represents intelligent action. There is no doubt that the medical profession is threatened with national legislation that, if enacted, would largely abolish medical practice as it is known today. Nothing is accomplished by the mere statement that a plan is "no good." Constructive criticism is required: in other words, those who are responsible for administering medical care and hence who are better informed concerning its practical aspects than are those not so engaged must point out *why* a scheme is unwise. In this way it is hoped that the desirable features of such plans can be promoted and that the undesirable ones can be avoided.

MEDICAL SOCIOLOGY

"Know thyself" is a wise command. For physicians, knowledge of themselves implies a judgment whether or not they are doing a good job in an imperfect world. For the individual physician, this judgment is easy to make regarding his technical work, but is difficult to appraise in relation to his equally important social functions. In the latter sphere, the facts are hard to come by, the objectives are poorly defined, and the opinions encountered are often based on emotion.

Physicians know that the world is changing at a breath-taking rate, and that the ways by which they serve the people need to be adjusted from time to time, if they are to continue to do a good job. Laymen are also aware of these facts and have been

giving increasing attention to a variety of programs designed to change existing arrangements between physicians and society. In times of rapid change it is even more than usually incumbent on physicians to know themselves, for theirs is the responsibility to direct the change in so far as it involves medical affairs. Physicians must continue to be educated, equipped, supported and stimulated to do a good job. To act intelligently they must know what kind of physicians America should have and in what kind of society these physicians are to work.

In these days when technical advance holds the center of the stage, most medical students have little opportunity to learn about either the relations between physicians and society or those between economics and disease. Few are able to approach the social or economic problems of the profession with the same intelligent attitude with which they face illness. Their education has been excellent from the technical point of view, but many of the fundamental problems besetting the profession of which they are about to become members have not been mentioned. Only a few students have interested themselves in these matters outside their studies.

Considerable impetus has been given to the teaching of such subjects by the Interim Report of the Social and Preventive Medicine Committee of the Royal College of Physicians of London.[1] Additional impetus has come from the establishment of a professorship of social medicine in Oxford University. An excellent definitive report has come from the latter source.[2]

As an experiment in teaching, in the winter of 1945 a series of nine lectures for third-year students was given under the auspices of the Department of Preventive Medicine of the Harvard Medical School, much of the stimulus having come from the students themselves. The first of these lectures was devoted to the broad problem of the individual as a social being and to the effect of social and environmental factors on his health. The material in this lecture is considerably augmented by work on individual patients in the final medical-school year. The remaining eight lectures were given as a series on medical sociology. The lectures were concerned with the study of physicians and the medical profession in relation to society. Forces determining the past, present and future structure of medical practice were considered. The obligations and responsibilities of the medical profession to society were brought clearly into focus. The speakers at all but the last three lectures were requested to present factual material and not their personal opinions or interpretations. It was thought that after having heard the factual data, the student would be in a position to reach his own conclusions, and to agree or disagree with the various opinions and points of view expressed in the final lectures. That some of the lecturers committed themselves as advocates of change is both natural in a free society and healthy for the future of medicine. The majority of the students and physicians attending the lectures expressed the opinion that they were helped in objectively facing the problems before them.

In view of the general interest to the medical profession of the problems presented and of the apparent success of this experiment in teaching, these lectures will be printed in consecutive issues of the *Journal*, beginning with this week's issue. It is hoped that they will be provocative of thought and interest on the part of physicians.

MASSACHUSETTS MEDICAL SOCIETY
POSTWAR LOAN FUND

The Postwar Loan Fund has been set up, and all discharged medical officers who were members of the Massachusetts Medical Society in good standing at the time of their entry into the service may apply for loans from this fund. For further information apply to:

George L. Schadt, *Chairman*
Postwar Loan Fund
8 Fenway
Boston 15, Massachusetts

REFERENCES

1. *Interim Report of the Social and Preventive Medicine Committee of the Royal College of Physicians of London.* 31 pp. London: Harrison & Sons, Ltd., 1943.
2. Ryle, J. A. Social medicine: its meaning and its scope. *Milbank Mem. Fund Quart.* 22:58-71, 1944.

MASSACHUSETTS MEDICAL SOCIETY

SUBCOMMITTEE ON MEDICAL ECONOMICS, POSTWAR PLANNING COMMITTEE

Information Concerning Recent Deliberations

The Subcommittee on Medical Economics met on September 13, 1945, to study Senate Bill 1050, the so-called "Wagner–Murray–Dingell Amendment to the Social Security Act."

It was soon obvious that before any critical study of this or any proposed legislation could be made, certain guiding principles on which all could agree should be formulated.

It was also pointed out that Senate Bill 1318, the so-called Pepper "Maternal and Child Welfare Act of 1945," might soon come up in committee and that a study of this bill should take precedence over that of Senate Bill 1050.

It was, therefore, agreed that the committee should:

(1) Outline the important basic principles that were considered essential to a successful medical-care plan.

(2) Prepare a brief discussion of the various methods of payment for medical services.

(3) Study and prepare a critical report on Senate Bill 1318.

Subsequent meetings were held on September 27, October 18, November 1, 2, 4 and 19 and December 10 for periods varying from five to seven and a half hours. Drs. Humphrey McCarthy, chairman of the Committee on Legislation, Joseph O'Connor, of Worcester, Stewart Clifford, of Boston, and Caroline Chandler of the State Department of Public Health took an active part in the discussions that followed and were most helpful to the subcommittee. What at times appeared to be widely divergent attitudes were, after free discussion, molded into this report.

The following basic principles were evolved as being essential to a medical-care plan in a free society:

(1) The objective of adequate medical care in our free society is to make available to everyone — regardless of race, color, creed, financial status or place of residence — every known essential preventive, diagnostic and curative medical service of high quality. The attainment of such medical care must necessarily be an evolutionary process that will require the co-operation of all concerned over a period of years.

(2) The success of any plan for medical care is dependent on the mutual co-operation of the public, those rendering professional services and the administrative agencies. This co-operation can be obtained only if those rendering the services are convinced that they will have a continuing authoritative voice in the formulation and execution of policies and plans, thereby assuming their proper share of responsibility.

(3) Provision of adequate medical care for those unable to obtain it by voluntary prepayment plans or by direct payment is the responsibility of the local or state government. Part of the burden of this responsibility may be assumed by charitable agencies. Federal grants-in-aid to state programs administered by state boards of health are an acceptable method of helping to meet this responsibility.

(4) The medical care of those who are able to purchase it by voluntary prepayment plans or by direct payment is the responsibility of the individual.

(5) Eligibility for receiving benefits under a program aided by federal grants should be determined by the individual states.

(6) The patient shall have free choice of his physician, group of physicians, clinic or hospital from among those participating in any plan, provided that the physician, group of physicians, clinic or hospital selected shall have the right to refuse to accept the patient.

(7) Physicians and other qualified persons rendering medical care should receive adequate remuneration for their services. Eventually this should include payment for services rendered to needy patients both in and out of hospitals.

(8) The physician shall be free to elect or reject without prejudice participation in a medical-care plan. The rights of the physician as to the choice of methods by which he is to be paid shall be fully protected.

Methods of Paying Physicians Under Medical-Care Plans

In considering the various methods of paying physicians under such plans, several points must be kept clearly in mind to avoid drawing misleading conclusions from the experience of private practice.

First, in private practice, where the patient pays the bill, there is a deterrent to either excessive demands by the patient or excessive services by the doctor. In any plan where a third party pays the bill, this deterrent is largely removed regardless of the method of payment. Under medical-care plans, provisions against excessive demands should be made by specific clauses in the contracts or by establishing regulatory measures.

Second, under a medical-care program where patients are free to choose their physicians and doctors are free to accept or refuse individuals as patients, the stimulus to the patient and the doctor for establishing a satisfactory relationship is almost independent of the method of payment.

Third, the same opportunity for group practice pertains under any method of payment.

Incidentally, the subcommittee reaffirmed its approval of the Blue Shield prepayment plan spon-

sored by the Massachusetts Medical Society, as stated in the September 20 issue of the *New England Journal of Medicine*.

Fee-for-service. Under a fee-for-service system, practitioners, specialists and consultants are paid a stated value for each unit of service rendered.

In current plans the physician is required to submit to the administrative agency a clinical report and a bill or voucher for each service rendered. This entails the administrative costs of handling such reports and vouchers and may require the administrative agency to set up a system of cross index concerning the practices of each physician and the health of each patient.

When a doctor cares for few patients under a medical-care plan, fee-for-service provides a remuneration that is proportional to the service rendered and thus covers the uneven incidence of illness among such patients.

Under any system of payment, fee-for-service is an appropriate way to pay for consultant or for emergency services not rendered by the patient's family doctor.

Under medical-care plans, experience shows that this method of payment is susceptible to abuse by a small percentage of physicians and patients. If the total budget of the medical-care plan is limited and demands are in excess of the funds, the payment per unit of service may be reduced.

Per capita. The per-capita system applies primarily to general practitioners. Under this system, practitioners are paid a certain sum per patient per year for each patient who elects him and whom he accepts. This payment is made regardless of the amount of care given to different patients or the fact that some patients will receive no service. A doctor being paid on this basis may be paid in addition for consultant or unusual services on a fee-for-service or per-case basis.

The practitioner is required to notify the administrative agency when he first becomes the practitioner of a given person. This provides the administrative agency with the necessary information for remuneration of physicians. The administrative agency may from time to time request additional information from the doctor for study or other informative purposes. Physicians are usually given the choice of receiving remuneration quarterly, semiannually or annually.

When a doctor cares for enough patients under a medical-care program to provide an actuarily sound distribution of risk due to the uneven incidence of illness among his patients, the per-capita method of payment permits economy in administrative burden with equity to the doctor.

This method of payment is not susceptible to excessive care by the practitioner but is susceptible to excessive demands by the patient.

Salary. Under the salary method, a stipulated compensation for services rendered is regularly paid a physician, usually per week or per month.

For certain physicians practicing as members of well organized groups or institutions, this form of payment provides a graded and predictable remuneration.

It may provide the means of attracting a physician to a rural community to which he otherwise would not go because of irregularity of income.

If groups of physicians or institutions contract to provide medical care either as consultants, specialists or practitioners, salary may in certain instances be a desirable form of paying for the medical care so provided.

If provision is made on a merit basis for advancement and increased salary, the competition for high-salaried positions provides the incentive to a high quality of endeavor on the part of physicians, just as it does in other professions and occupations.

This method of payment necessitates but little administrative paper work.

Per case. Under the per-case method a physician is paid a stipulated sum for the total service he renders a patient during a particular illness or because of a particular condition, usually within a stated time.

It is particularly applicable to consultant or specialist services rendered to certain types of cases.

Under the per-case basis a single clinical report and a bill are usually required to be submitted only at the termination of the services rendered.

Per session. The per-session method applies a flat rate of payment for the services rendered per stipulated session.

Payment on this basis is particularly applicable to services rendered intermittently in group clinics.

It usually requires the submission of a report of the physician's presence at the session.

Cash indemnity. Under the cash-indemnity system, payments are made by the administrative agency to the patient, not to the doctor. It provides for payment of a flat sum for wages for time lost because of illness or cash payments toward the cost of medical care according to a fee schedule, or both. Each person is free to make his own arrangements with those who furnish medical services as to both the amount of service and the charges for it.

It provides no assurance that the money paid to the patient will be expended on medical care, and little control over quality or cost. Such supervision as would check on the expenditure of the funds for the medical care for which they were allotted and on the cost and quality of the care provided would entail an almost insurmountable task.

Physicians might be obliged to spend time on examination for certification of illness to permit

the patient's collection of a cash indemnity rather than for the treatment of illness.

Although the amount of administrative paper work concerning medical care would be small, that required by the professional certification of illness might be great.

The Maternal and Child Welfare Act of 1945

The Maternal and Child Welfare Act of 1945 (Senate Bill 1318) has been carefully studied by this subcommittee. Although this subcommittee is in complete agreement with the objectives stated in the introduction to the bill, — "To provide for the general welfare by enabling the several states to make adequate provision for health and welfare of mothers and children and for services to crippled children," — it finds serious objections to the bill as written. Some of the more important of these are as follows:

(1) The bill makes no adequate provision for general public-health programs that are more fundamental than this specialized legislation.

(2) Services and facilities are available to all who elect to participate, regardless of economic status. This violates Basic Principles 4 and 5.

(3) The public deserves a reasonable estimate concerning the ultimate cost of this proposed legislation. Experience and such factual data as are available indicate an ultimate annual budget of approximately one billion dollars. This should be clearly recognized in any consideration of the bill.

(4) Fee-for-service method of payment is restricted to consultation or emergency visits and is not ordinarily available to practitioners or specialists.

(5) The bill does not make clear just who is to decide the fee for a given service that would be considered adequate remuneration in an individual state, nor does it make provision for variable fees to meet the differing costs in the several states.

(6) The bill does not prohibit professional personnel, groups or institutions rendering service under the program from accepting supplemental payment from or on behalf of patients.

(7) The bill does not provide for payment to groups of physicians, clinics or hospitals providing professional services.

(8) The bill does not emphasize the desirability of full utilization and further development of existing services and facilities.

(9) The bill does not emphasize the necessity of restricting the development and expansion of a state program to the capacity of available administrative and professional resources.

(10) The bill does not provide the professions rendering service a continuing and authoritative voice in the formulation of policies and plans. Such committees as are selected under the provisions of the bill may represent the attitude of the administrator rather than that of a given profession or group. This violates Basic Principle 2.

(11) The bill does not provide the facilities whereby the opinions of both the federal and state advisory committees are made available to the public.

(12) Designation of the Children's Bureau as the administrative agency does not adequately assure proper integration of the health activities of the federal government.

For these reasons we believe that the Maternal and Child Welfare Act of 1945 (Senate Bill 1318) does not represent the best form of legislation for the purposes for which it was written.

LELAND S. MCKITTRICK, *Chairman*

MISCELLANY

NEW SURGICAL HEAD AT BOSTON UNIVERSITY

Dr. Reginald H. Smithwick has recently been appointed professor of surgery and chairman of the Department of Surgery of Boston University School of Medicine and surgeon-in-chief of the Massachusetts Memorial Hospitals. Dr. Smithwick was formerly associate visiting surgeon at the Massachusetts General Hospital and instructor in surgery at Harvard Medical School. Dr. Smithwick, who is well known for his original contributions to the study and surgical treatment of hypertension and peripheral vascular diseases, will continue and extend his investigations. In this work he will have the close collaboration of the members of the faculty of the Boston University School of Medicine who have been engaged for several years in independent investigations of problems that are closely allied to Dr. Smithwick's studies.

NOTICES

ANNOUNCEMENTS

Dr. Carlyle G. Flake announces the reopening of his office at 300 Longwood Avenue, Boston, for the practice of diseases of the ear, nose and throat.

Dr. Henry A. Kontoff, having returned from active service with the United States Navy, has resumed the practice of urology at 483 Beacon Street, Boston.

Dr. Walter S. Levenson, having returned from active service with the United States Navy, has resumed the practice of general surgery at 370 Commonwealth Avenue, Boston.

Dr. Abraham Wolbarsht, having returned from active service with the United States Army, has resumed the practice of internal medicine at 375 Commonwealth Avenue, Boston.

SOUTH END MEDICAL CLUB

The next regular meeting of the South End Medical Club will be held at the headquarters of the Boston Tuberculosis Association, 554 Columbus Avenue, Boston, on Tuesday, January 15, at twelve noon. Dr. Frank A. Pemberton will speak on the subject "The Significance of Bleeding from the Female Genital Tract." Dr. D. D. Hall will preside.

Physicians are cordially invited to attend.

(*Notices continued on page xv*)